THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—18TH YEAR. SYDNEY, SATURDAY, OCTOBER 31, 1931. No. 18.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.

"Some Aspects of Diabetic Coma and Arteriosclerosis," by L. W. DUNLOP, M.B. 533
"Typhoid Carriers: Observations on Their Investigation and Treatment," by A. R. SOUTHWOOD, M.D., M.S., E. B. THOMAS, M.B., B.S., and R. B. KNIGHT 541
"Affections of Peripheral Nerves," by A. W. CAMPBELL, M.D. 544
"Affections of the Peripheral Nerves," by J. C. B. ALLEN, M.B., Ch.M., F.R.C.S. 546

REPORTS OF CASES—

"Thyreoid Disease," by GEOFFREY A. PENINGTON, M.D. 550

REVIEWS—

Exercise for the Middle-Aged 552

LEADING ARTICLES—

The Wassermann Test 553

CURRENT COMMENT—

Circulatory Mechanism of Marathon Runners .. 554
John Monash 555

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Gynæcology 556
Obstetrics 557

SPECIAL ARTICLES ON AIDS TO DIAGNOSIS—

The Clinical Significance and Interpretation of the Wassermann Reaction 558

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 560
Nominations and Elections 564

OBITUARY—

Robert Gordon Craig 564
Thomas Howard Morgan 566
John Monash 566

CORRESPONDENCE—

Orthopædic Surgeons 566
The Diagnostic Use of Tuberculin 567

DIARY FOR THE MONTH 568

MEDICAL APPOINTMENTS VACANT, ETC. 568

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 568

EDITORIAL NOTICES 568

SOME ASPECTS OF DIABETIC COMA AND ARTERIOSCLEROSIS.[1]

By L. W. DUNLOP, M.B. (Sydney),
Honorary Physician, Sydney Hospital.

DIABETIC COMA.

DIABETIC coma first appeared in the literature as a separate entity in 1854. A masterly description was published by Kussmaul, of Strasburg, in 1874; he described the characteristic breathing, the outstanding feature of the condition, which still bears his name.

In order to get an idea of the incidence and mortality of this condition I have investigated the case records of diabetes in Sydney Hospital from 1922 to 1931. In this period 528 patients with diabetes were admitted as in-patients; 45 suffered from coma and of these 27 died. In the period January, 1922, to August, 1926, there were 200 patients, of whom 22 suffered from coma. All of these coma patients died. This 100% mortality appears partly due to the fact that insulin was not used before August, 1923, and partly to the number of obviously moribund patients admitted. Patient number 381 was in hospital only two hours before death,[1] while nine of the 27 patients who died averaged only four and two-third hours in hospital.

Another factor early in this period was expense and irregular supply of insulin. The case notes of patient number 49, who died in September, 1923, indicate irregular supply, and a final note states: "This patient, during seven weeks' treatment, has cost in insulin £19 11s."

The results show considerable improvement in the second period, August, 1926, until July, 1931. Diabetic patients numbered 328. Of these 23 could

[1] Read at a meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association on September 17, 1931.

[1] The numbers throughout this paper are those of the Sydney Hospital records.

be considered as suffering from coma. Five died, giving a mortality rate of 21·7%. During the last two years there were five cases of coma; all the patients recovered.

Comparison with figures in the literature is interesting. H. J. John, writing in 1929, showed that the average mortality rate among cases reported at that time was about 25%. These figures are shown to you in tabulated form. He also stated that before insulin was used, the mortality rate in coma was practically 100%.

Statistics of 2,572 diabetics at the Cleveland Clinic showed that 71 patients with coma were treated personally by him; of these four died, a mortality rate of 5·6%. A further 32 patients with coma belonging to this series were "treated elsewhere"; 28 died, a mortality rate of 87·5%.

The Sydney Hospital series of 528 diabetics from 1922 to 1931 have been considered in two periods.

In the first period, January, 1922, to August, 1926, 200 diabetics were treated; 22 suffered from coma and these 22 died, a mortality of 100%.

In the second period, August, 1926, to July, 1931, 328 diabetics were treated; 23 suffered from coma and of these five died, a mortality of 21·7%.

Ætiology.

When carbohydrate oxidation is seriously impaired, fat metabolism becomes abnormal, ketone bodies, oxybutyric acid, diacetic acid and a derivative—acetone—accumulate, and coma may follow. This is the so-called non-oxidation theory, and is well expressed in the metaphor: "Fat burns in the flame of carbohydrate."

In November, 1930, research by Chaikoff and Weber, and of Soskin at Toronto, was discussed in an editorial article in *The Journal of the American Medical Association*. These workers support another theory which assumes the conversion of fat into glucose. Mention of this appeared earlier in 1923, when Geelmuyden and other German workers advanced the view that insulin acts by inhibiting the over-production by the liver and other tissues of glucose and fat.

Chaikoff and Weber gave repeated injections of epinephrine to dogs from which the pancreas had been removed and from which food and insulin were withheld. They found an excretion of glucose far beyond what could be accounted for from preformed carbohydrate, protein and glycerine. A fatty acid source is assumed.

S. Soskin, writing in the *Journal of Nutrition* of September, 1930, expressed the opinion that if the demonstration that totally diabetic dogs can oxidize carbohydrate is valid, it can no longer be assumed that failure of carbohydrate oxidation causes disturbance in the utilization of fat. He thinks the ketones may be intermediate products in the conversion of fat into glucose, and that their accumulation, like that of dextrose, is due to excessive gluconeogenesis. This has been called the "over-production" theory.

Diabetic coma is usually regarded as due to acidosis, caused by the accumulation of these acid bodies, the ketones in the organism, and the consequent depletion of the alkali reserve. This theory is supported by the evidence of definite lowering of the carbon dioxide tension of the alveolar air and alkali reserve which is present in most cases. Some observers, however, consider the condition is more in the nature of a poisoning or ketosis, due to an enolic group in diacetic acid. The question was dealt with recently by Dodds and Robertson in a paper published in April, 1930.

They investigated a series of coma cases and stated that "observations seem to show that neither the acidosis nor ketosis theory adequately explains the cause of diabetic coma." They cite a fatal case in which the aceto-acetic acid content of the blood fell steadily until the fatal termination, while the alkali reserve rose. The problem is obscure, and it is difficult to say in the light of our present knowledge which is the correct view.

The chief factors precipitating coma are: (i) omission of insulin, (ii) indiscretions of diet, (iii) infections.

Omission of Insulin.

If a diabetic is unable to eat owing to different causes, such as gastro-intestinal lesions, infections *et cetera*, he often omits taking insulin. He or his family may have been taught to fear the risk of hypoglycæmia at such times. He should be told that when he does not eat, the body burns or eats its own tissues and for this insulin is essential if coma is to be avoided.

H. J. John wrote in 1928: "The unfortunate idea that a patient who is not eating must not take insulin is still in vogue."

In the Sydney Hospital series patient number 314 felt sick, could not eat, and omitted two doses of insulin. She vomited and in a few hours was comatose.

Joslin warns diabetics taking insulin that if they omit it and sugar appears in the urine, they are in danger of coma. Patient number 333 was a case in point. She left hospital on diet and insulin and was readmitted shortly afterwards in coma. She was unable to afford insulin and had been "eating anything."

Indiscretions of Diet.

Indiscretions of diet are the main cause. In Joslin's series 70% of cases gave this history. As a rule nausea and vomiting ensue, and acidosis quickly develops.

In Sydney Hospital series patient number 250 "ate too much at a party" and was admitted next day in coma. Patient number 326 "had a feed of biscuits" and was admitted soon after, unconscious.

Infections.

Infections that precipitate coma are often not severe and are frequent in the upper respiratory tract, tonsillitis, *otitis media et cetera*. Coma is generally slow in developing, and there is time as a

rule for preventive measures. Acute panophthalmitis was the factor in patient number 97 of our series, and in patient number 321 acute balanitis and paraphimosis were responsible.

Other Causes.

A fatal case occurred early last year as a result of defective Benedict's solution.

A diabetic was admitted under my care in the Mater Misericordiæ Hospital suffering from failing heart and severe acidosis. Insulin and digitalis were given and the patient improved. On the third day, according to tests with Benedict's solution, she was aglycosuric, and insulin was not given. Next day she was worse, and Fehling's as well as Benedict's test was applied. The former was strongly positive, the latter gave no reaction. In spite of resumption of insulin she died in coma a few hours later.

Allan and Wilder, writing from the Mayo Clinic in January, 1930, cite a similar case and state:

It is not uncommon for patients to obtain improperly prepared Benedict's solution. Sometimes the quantitative is dispensed instead of the qualitative solution.

They teach patients to check the solution before use with a drop of honey or corn syrup. Other similar instances have occurred in Australia. Personally I have always preferred Fehling's solution.

A long train journey was apparently the responsible factor in the first attack of coma of patient number 200. In others accidents or mental stress may operate.

Symptoms and Signs.

The onset may be rapid. John cites one of Van Noorden's cases. A bank director in apparent good health returned home after a stormy meeting; later, after dinner, he was found on the sofa in coma.

As a rule the onset is gradual and the early symptoms are frequently misleading. The development of dehydration is the outstanding feature of the condition. As acidosis develops, the patient experiences nausea, abdominal pain and may vomit; later he becomes drowsy.

As a result less and less fluid is taken; in the first place because of nausea, later because he is too drowsy to heed his thirst. In addition glycosuria and polyuria are operating, and air hunger is now added to help in water loss from the lungs. The net result is dehydration of the tissues, which is seen in the dry, parched tongue, sunken and often soft eyeballs (Krause's sign) and loose skin. Drowsiness increases and Kussmaul breathing becomes prominent. In this type the inspiration is loud and prolonged, followed after a pause by short expiration. The breath has an odour of acetone. The temperature is usually subnormal, but may be raised in the presence of an infection. The pulse is fast and the blood pressure low, owing to cardiac weakness and dehydration. The vomit may contain blood and the patient becomes unconscious, with flaccid muscles, the knee jerks disappearing as a rule early.

The urine is in some cases diminished in quantity or suppressed, while sugar, abundant diacetic acid and acetone are present.

Albumin, casts and red cells often appear in the urine, as the kidneys bear the brunt of excreting the acid bodies. The blood shows hyperglycæmia and often lipæmia, while the carbon dioxide tension of the alveolar air is lowered.

Diagnosis.

The misleading character of early symptoms causes many errors.

Patient number 126 of our series, admitted in 1925, had been vomiting five days, and was sent in with a diagnosis of ptomaine poisoning. He died five and a half hours later.

A patient, admitted on June 26, 1930, under my care in the Mater Misericordiæ Hospital, was sent in on account of vomiting, with provisional diagnosis of intestinal obstruction. She was found to be suffering from early coma, and received 300 units of insulin in twenty-four hours before recovery.

When the patient is comatose diagnosis is not always easy. Hypoglycæmic coma must always be excluded. Always inquire if insulin has been taken beforehand. Get a specimen of urine. The absence of sugar, the quiet breathing, the moist tongue and skin of typical hypoglycæmic coma are in marked contrast to the noisy breathing, dry tongue and generally desiccated look of typical diabetic coma. The administration of sugar quickly decides the question.

Cerebral conditions may be mistaken for coma.

One evening early last year I was called to see a patient in consultation, thought to be suffering from diabetic coma or hypoglycæmia. He was restless and semiconscious, but could not answer questions. The breathing was quiet, the skin moist and there was no odour of acetone. The pupils were unequal, and he had spasticity and an extensor plantar reflex on one side. A catheter specimen contained sugar, but no acetone or diacetic acid. (He had been given glucose.) His wife then arrived at the hospital, and she gave his history as one of gradually increasing headache and mental dulness for some weeks. He had been taking insulin for some time longer. Lumbar puncture was done, and clear fluid issued under considerable pressure. He quickly became conscious. Next day he was free from headache, and the extensor plantar reflex and spasticity had disappeared. Later optic atrophy was found, and X ray examination disclosed enlargement of the *sella turcica*. He finally died. No doubt a tumour in the region of the pituitary gland associated with diabetes existed.

Sugar and ketones are often present in the urine of patients suffering from cerebral lesions, but obvious neurological signs as a rule make the diagnosis clear.

A patient, E.T., aged sixty-four years, was admitted to Sydney Hospital in October, 1929, and died twenty-four hours after admission. In her case neurological signs were not obvious. She was deeply unconscious and dypnœic without cyanosis, with sugar and acetone in the urine. Two lots of forty units of insulin with glucose were given, and the blood sugar shortly after receded to 50 milligrammes per 100 cubic centimetres, so more glucose was administered. Before death the temperature rose to 41·6° C. (107° F.). Autopsy revealed a purulent meningitis.

In diabetic coma general muscular flaccidity and loss of knee jerks are signs of diagnostic importance.

Treatment.

As the early signs and symptoms are so often misleading, patients should be taught that whenever they feel ill or have fever, prophylactic measures must be adopted. They are briefly: to go to bed and

call a doctor, keep warm, take plenty of fluids and never to omit insulin if there is sugar in the urine.

When the diagnosis is established, the patient should be sent to hospital without delay, insulin being injected before he is moved, as there is practically no risk of hypoglycæmia in the first three or four hours.

Most of us are aware of and appreciate the necessity for hospital treatment, but it might interest you to know that as recently as 1928, H. J. John, writing in America on this subject, stated:

> Operations used to be done on the kitchen table in the home. Physicians are still living in the kitchen table era, but this period will surely pass and more persons will be saved.

Treatment in Hospital.

Good team work is of vital importance. The condition is desperate, the patient often literally struggling for life requires unceasing attention and activity on the part of both nursing and medical staff.

The patient should always have a special nurse and if possible should be placed in a special ward. In the general ward there is always the danger of the coma patient being treated merely as one of many, and the concern of no one in particular. For the first eight or twelve hours one nurse should be in sole charge, and should have her meals within call of the patient. She should be given full and written instructions.

In the large hospitals a resident medical officer, preferably a senior, should be deputed to take charge during the urgent period. He should establish liaison with the biochemists for the many blood sugar and other estimations necessary, be prepared to examine the frequent specimens of urine, and should see that apparatus for intravenous therapy *et cetera* and adequate supplies of glucose in distilled water solution are at hand. Only then, in the event of fatal termination, will we be able to say "everything possible has been done."

Insulin should be administered without delay and before any other measures are adopted. The main essentials of treatment are as follows: (i) insulin, (ii) fluids, (iii) glucose. General measures and cardio-vascular therapeusis are also necessary.

Insulin. Insulin administered in adequate doses quickly abolishes the excess of ketones, reduces the blood sugar and aids in replenishing the body glycogen.

In cases of moderate severity an initial dose of twenty to forty units is given subcutaneously and repeated every half hour, every hour or every two hours. Patients in deep coma should be given fifty to one hundred units intravenously, and this may be repeated three or four times in the first twenty-four hours. Subcutaneous injections are often combined with intravenous medication; the effect is thought to be more lasting. Usually within twenty-four hours there is striking improvement, and the insulin may be reduced to ten to twenty units three or four times a day. There is no exact dosage of insulin in the treatment of coma. In the first twelve to twenty-four hours the patient may require anything from one hundred units to one thousand units or more.

A warning is necessary in coma due to infections. Not only should an acute infection, such as *otitis media* or appendicitis, not be overlooked, but it should be remembered that when an infection with elevation of temperature is present, the patient is resistant to insulin and may require much larger doses.

Danger occurs also when the patient commences to recover from the infection. This may arise almost at any moment, and what was previously an adequate dose becomes an overdose. Alarming symptoms of hypoglycæmia may quickly develop.

Fluids. At least four litres of fluid in twenty-four hours for the first two or three days should be got into the patient by mouth, stomach, tube, *per rectum*, subcutaneously or, in extreme cases, intravenously. Dehydration is always present and must be vigorously countered. Half a litre or more of saline solution under the breast at the outset is always beneficial.

Glucose. Some authorities give glucose with insulin in every case, whether or not there is blood sugar control. Joslin advises against it. As long as there is adequate blood sugar control, glucose would not appear to be necessary until the blood sugar is reduced to 250 milligrammes. It is then given in conjunction with the insulin, one gramme for every unit.

Where blood sugar estimation is not possible, glucose (one gramme to one unit of insulin) is always given to avoid hypoglycæmia. In these cases a sample of urine should be obtained before each injection of insulin, and should show sugar in small amounts.

In desperate cases when intravenous medication is adopted, the glucose may be advantageously combined in 10% solutions with the insulin that is injected; 250 cubic centimetres is a quantity that is often given, and a cardiac stimulant, such as caffeine sodio benzoate, 0·3 to 0·4 gramme (five to six grains) is often added.

General Measures. The temperature is usually subnormal and the pulse feeble. Warmth and the usual measures to combat shock and collapse are essential and should be attended to as soon as the initial dose of insulin is given.

For cardiac symptoms digitalis or caffeine sodio benzoate is useful, while a coffee and brandy enema is usually of great benefit.

Vomiting is usually present. The stomach should be washed out unless the patient is very collapsed. The bowels should be emptied by enema, but purgatives are to be avoided for fear of aggravating dehydration.

When the urine is free from ketones and the patient is obviously recovering, a normal diet should be gradually resumed. Until then no food is necessary; glucose and fluids are quite adequate for the condition.

Alkalis in treatment have not been used to any great extent for many years. References have recently appeared in the literature, however, indicating that they are again finding favour in certain cases.

Time is the essence of the contract in treatment of coma. These cases are just as urgent as acute general peritonitis, and even a delay of half an hour in beginning treatment may prove fatal.

A good example of the fulminating nature of coma occurred in my experience as recently as August 6 last.

On the morning of August 6 a middle-aged diabetic who had had diarrhœa for two days went to work in the city. Feeling very ill, he returned to his home in North Sydney about midday, and shortly after became unconscious. He was admitted to hospital by his doctor, who gave him fifty units of insulin intravenously soon after 2 p.m.; a previous blood sugar reading was 300 milligrammes. Further intravenous insulin (fifty units) was given about 5.30 p.m., but he was still deeply unconscious. I arrived at the hospital for consultation about 7.40 p.m., to find that he had died a few minutes previously.

From the description of the signs present I have no doubt that he was suffering from typical diabetic coma, but in spite of prompt treatment he was overwhelmed and died in a few hours.

The following two case records illustrate some points of interest.

Patient number 200, E.W., a female, aged seven, was admitted to hospital in December, 1923. She had just arrived by train from Mudgee and was in a comatose condition. On examination she was emaciated and restless, with marked air hunger and extreme dehydration; the temperature was subnormal, and the urine was loaded with sugar, acetone and diacetic acid; she could be roused with difficulty.

Insulin was not available. She received intravenous and subcutaneous injections of saline solution and copious fluids by mouth and rectum, with glucose and oatmeal gruel by mouth. Caffeine sodio benzoate was also given. After two days' treatment she recovered. A day or so later she developed œdema, which disappeared when salt was withheld.

She was admitted to Sydney Hospital on June 4, 1924, for further treatment and remained there until June, 1926.

On admission weight was 21·48 kilograms (three stone five and three-quarter pounds), her height was 117·5 centimetres (three feet eleven inches). Two years later her weight was 27·4 kilograms (four stone five pounds) and her height about 130 centimetres (four feet four inches). Insulin treatment was commenced during June, 1924, and she received an amount of 20 to 30 units every day during this period in hospital.

After treatment with insulin for some months an interesting condition was found, namely, hyperglycæmia associated with raised renal threshold. This existed apparently without cardio-vascular or renal complications.

On June 5, 1924, she had a fasting blood sugar of 130 milligrammes, and sugar was present in the urine.

On June 14, 1924, a glucose tolerance test revealed: after a half hour interval, blood sugar 182 milligrammes; after one hour, 375 milligrammes; after one and a half hours, 565 milligrammes; after two hours, 550 milligrammes; 1·76% sugar was in urine for the previous twenty-four hours.

On November 7, 1925, the fasting blood sugar was 269 milligrammes, there was no sugar in the urine; three hours after a meal the blood sugar was 340 milligrammes; there was no sugar in the urine.

On January 21, 1926, the fasting blood sugar was 203 milligrammes, there was no sugar in the urine; three hours after a meal the blood sugar was 254 milligrammes; there was no sugar in the urine.

On March 1, 1926, the blood sugar was 271 milligrammes, the urine contained no sugar.

On May 30, 1925, after 20 grammes of glucose, the figures were 354 fasting, 453, 351, 532, 495.

This phenomenon is reported in the literature. Rabinowitch reports fourteen cases in which, after the use of insulin for a varying period, the threshold rose materially. Before insulin was given these patients showed an average blood sugar of 180 milligrammes, with glycosuria. After insulin treatment for some months the average figure for blood sugar was 290 milligrammes, with no glycosuria. There was no evidence of impaired renal function.

Major and Davis, from the Department of Internal Medicine, University of Kansas, reported eight similar cases in 1925. In several of these cases attempts to lower the blood sugar by means of insulin resulted in mild insulin shock.

It is possible that similar symptoms arose in my patient when she received large amounts of insulin at a later date for coma.

She was sent to Canonbury Convalescent Home in June, 1926. On August 4, 1926, at 9.30 p.m., she was brought back to Sydney Hospital in coma, as a result of overeating. Similar lapses had occurred with production of severe acidosis on several occasions during her previous stay in hospital. On admission her condition was grave. She was practically unconscious, but could be roused just enough to grunt unintelligibly. Her condition during seventeen hours is set out in Table I.

This child received 280 units of insulin in seventeen hours, and apparently died from sudden cardiac failure. This is a frequent cause of death in coma. Joslin quotes Strauss, who observed during the world war that many diabetics died in coma from failure of the circulation rather than from the coma itself.

In the second case an infection was apparently responsible for the patient's condition.

Could death have been in part due to insulin shock, as already mentioned when discussing the high renal threshold in this patient?

H.C., a male, aged seventeen years, patient number 321, was admitted at 5 a.m. on May 23, 1928. He was seen two days previously and admission was recommended; blood sugar then was 691 milligrammes. An infection was apparently responsible for his condition. Details of his treatment are shown in Table II.

During the first fourteen hours of treatment 235 units of insulin were given. During the next twenty-four hours he had fifty units of insulin, plenty of fluids, some milk and glucose. Blood sugar estimations were 364 milligrammes, 480 milligrammes and 465 milligrammes, 250 milligrammes at 9.30 a.m., 2 p.m. and 12 midnight.

Arteriosclerosis.

Since the advent of insulin, arteriosclerosis has become a subject of great interest in diabetes. As a result of improved treatment, diabetics live longer, and so are more likely to develop and die of vascular disease. Joslin writes:

No one worried about the development of old age or arteriosclerosis in the diabetic a generation ago, but today this is the foremost factor in the treatment of the disease, because approximately one-half of those who have diabetes suffer with and ultimately succumb to arteriosclerosis.

This condition has unexpectedly developed into a problem of major importance in diabetes, and our

Table I.

Hour.	Condition.	Urine.			Blood Sugar.	Treatment.
		Sugar	Acetone	Diacetic Acid		
0	Air hunger, dehydrated, acetone odour. Temperature 35·1° C. (95° F.), pulse 104, respirations 28. Vomited the glucose by mouth.	+++	+++	+++		Insulin 20 units, glucose 20 grammes. Rectal salines, 5% glucose, 3-hourly, strychnine and digitalin 4-hourly; warmth; special ward and nurse; enema.
1	Glucose retained by mouth.					Insulin 20 units, glucose 20 grammes.
2	Temperature 35·1° C. (95° F.), pulse 132, respirations 32.					Insulin 20 units and glucose 20 grammes by mouth.
3						Ditto, warmth applied.
4	Vomited brown fluid, passed urine.	+++	+++	+++		Insulin 20 units, hypodermic injection.
5	More conscious, sucked orange, seemed better.					Insulin 20 units, hypodermic injection.
6	Temperature 36·1° C. (97° F.), pulse 130, respirations 38. Orange given.					Insulin 20 units, hypodermic injection. Half hour later vomited green fluid.
7						Insulin 20 units hypodermic injection; strychnine and digitalin.
8	Not so well, improved after intravenous injection.					Insulin 20 units, hypodermic injection; 10 ounces of 5% glucose and 20 units of insulin given half hour later intravenously.
10	Has been vomiting.					Insulin, 20 units, hypodermic injection, and water to drink.
11	Quite conscious and answers alertly. Ordered caffeine sodio benzoate, 3 grains.	++	++	++	542 milligrammes.	
12	Temperature 36·1° (97° F.), pulse 130, respirations 36.					Insulin, 20 units, and 5% glucose intravenously; saline solution given by the rectum.
13						Insulin 20 units, glucose 20 grammes intravenously.
14						Insulin 20 units, glucose 20 grammes intravenously; caffeine sodio benzoate given half hour before.
16	Much better, taking plenty of fluids.					Insulin 10 units, hypodermic injection.
17	Much worse; pulse fast, 160; temperature 35·6° C. (96° F.); signs of œdema in lungs. Died suddenly ten minutes after insulin. Signs of acute heart failure.				293 milligrammes half hour before death.	Atropine 0·54 milligramme ($^1/_{120}$ grain) and caffeine sodio benzoate given. Insulin 10 units intravenously.

Table II.

Hour.	Condition.	Urine.			Blood Sugar.	Treatment.
		Sugar	Acetone	Diacetic Acid		
0	Semiconscious, air hunger marked, acetone odour, dehydration, acute balanitis and paraphimosis.	+++	+++		512 milligrammes.	Insulin 50 units intravenously; paraphimosis reduced; enema and fluids given.
2						Insulin 40 units intravenously half hour previously.
5					357 milligrammes.	Insulin 40 units, hypodermic injection; by mouth glucose, orange and water.
7						Insulin 30 units, hypodermic injection.
10					293 milligrammes.	Insulin 15 units, hypodermic injection half hour previously; glucose 5 ounces 20% every four hours *per rectum*.
12						Insulin 30 units, hypodermic injection; glucose by mouth.
14	Much improved.				189 milligrammes one and a half hours later.	Insulin 15 units, hypodermic injection.
18						Insulin 15 units, hypodermic injection.
19	Recovered from coma symptoms.	++	—	—	226 milligrammes.	Orange juice every four hours, four ounces.

recorded experience in Australia is small. I trust, therefore, you will excuse what must be of necessity largely a review of the overseas literature on the subject.

Incidence of Arteriosclerosis.

Evidence of the incidence of arteriosclerosis is obtained by consideration of the following factors: (i) analysis of cause of death, (ii) autopsy findings, (iii) clinical and physical examination, (iv) X ray examination of the vessels of the extremities.

Analysis of Causes of Death. The analysis of 1,756 deaths among cases of true diabetes treated by Joslin, shown in Table III, is instructive.

Deaths from arteriosclerosis include those from chronic cardiac and nephritic disease, from apoplexy, gangrene and sepsis (extremities), as well as from uncomplicated arteriosclerosis. Coma as a cause of death has fallen from 61% in the early period to 10% at the present time. Arteriosclerosis has advanced from 15% to 47%. It is also apparent that diabetics now live longer and the duration of the disease is longer.

Autopsy Findings. In Joslin's series 52 cases were examined. Diabetes had lasted less than five years in 33 instances. Arteriosclerosis existed in 70%. Diabetes had lasted over five years in the remaining 19 cases. All of these showed arteriosclerosis (100%).

Clinical and Physical Examination. Clinical evidences of vascular changes are to be found in the brain, heart and kidneys. The radial arteries may be studied by palpation, and ophthalmoscopic examinations are also of great value.

X Ray Examination. X ray examination of the vessels of the extremities, as will be noted later on, also affords valuable evidence.

Ætiology and Pathology.

It might be said that the incidence of arteriosclerosis in diabetes is due solely to old age and increased duration of disease. Evidence, however, is accumulating that other factors exist. These will now be considered in conjunction with the pathology.

The Age Factor.

Shepardson, in a recent paper (May, 1930), published results of investigation in a group of fifty diabetics of an average age of 23·4 years and with an average duration of disease of 6·9 years. Evidence of vascular sclerosis in the extremities, as shown by X rays, was found in 18 cases (36%). The average duration of disease, 6·9 years, he considered only of importance because the causative factor must act over a reasonable period of time before its effects became manifest.

Priscilla White, writing in October, 1930, discussed the incidence of arteriosclerosis in diabetic children. In 11 children, all of whom had diabetes in the pre-insulin period, X ray evidence of vascular changes, namely, calcification of the vessels of the legs, existed. Further, a group of 59 children, treated with insulin for seven years, were selected and ten were examined by X rays. None showed vascular sclerosis. This result, she states, should be compared with the incidence of vascular changes in one out of three pre-insulin cases studied. "In our experience," she remarks, "it is the diabetic child, treated with the inadequate methods of yesterday, who has developed cataracts and arteriosclerosis."

Bowen and Koenig, in X ray studies published in 1927, report a special incidence of arteriosclerosis in diabetes. Arteriosclerosis was found in 63% of a group of diabetics above the age of forty, while in a group of non-diabetics of similar age the disease was present in only 28%.

They, too, found that arteriosclerosis appeared mostly in diabetes of long standing when treatment had been neglected.

Faulty Fat Metabolism and Diet Factors.

Aschoff, who has reaffirmed and amplified Virchow's imbibition theory, attributes arteriosclerosis to the deposit of fat in the form of cholesterol esters in the arteries. The greater the amount of fat in the blood, the more readily is it deposited. This applies particularly to patients with severe or neglected diabetes, who generally show evidence of acidosis and increased blood fat.

Joslin is also in favour of this and states:

> I believe the chief cause of premature development of arteriosclerosis in diabetes, save for advancing age, is due to excess of fat in the body (obesity), in the diet, and in the blood.

He blames high fat diet and more especially those diets rich in cholesterol. He cites a remarkable case reported by Gordon, Connor and Rabinowitch:

> A mild diabetic with onset at age of 45 years was treated for 13 years mostly on a diet low in carbohydrate and high in fat. His weight rose in five years before his death from 97½ lb. to 174 lb.

Some autopsy details are set out which are very similar to changes in the vessels as described by Virchow:

TABLE III.

Epoch.	Average Duration of Diabetes in Years.	Deaths Total.	Coma *per centum.*	Arteriosclerosis *per centum.*	Average Age at Death in Years.
Naunyn, 1894-1914	4·7	342	61	15	44·8
Allen, 1914-1922	5·4	805	42	26	
Banting, August, 1922, to July, 1926	7·7	609	20	47	54·2

The vertebral arteries contained large golden nodules. . . . . Sections from the vertebral, basilar and internal carotid arteries showed marked fatty infiltration. The lumina of some of the vessels were narrowed by the thickened intima and the large fatty cells beneath it. The media of some of the vessels was almost completely replaced by calcium and fat. The aorta showed marked fatty changes beneath the intima. . . .

The coronary vessels were markedly sclerosed. A section showed moderate intimal thickening and marked fatty infiltration beneath the intima. The femoral artery was involved.

Chemical analysis of the bright yellow portions of the vessels suggested the presence of vegetable lipochromes, in that they had high cholesterol contents.

Joslin comments:

With this portrayal before us, I doubt if ever again anyone will expose a diabetic to a low carbohydrate, high fat diet for so long a period.

Further support comes from Labbé and Heitz. They found cholesterol increased in the blood of 18 diabetics and 27 patients with *endarteritis obliterans*. They saw a close connexion between the cholesterol and the arterial disease.

Shepardson (previously quoted) is also in favour of faulty fat metabolism as a productive factor.

He found in a number of his group of fifty diabetics lower average blood cholesterol values than heretofore obtained, and parallel with this in the same patients a reduced incidence of arteriosclerosis. The lower blood cholesterol values he attributed to the use of insulin which acts by reducing the blood fat. He thinks that this association of lower cholesterol values and lowered incidence of arteriosclerosis necessitates the assumption that altered fat metabolism is a morbific factor in the development of vascular disease.

Apart from that due to ketones, acidosis in diabetes that may be related to diet has been considered as a causal factor. Workers at the Potter Memorial Clinic, Santa Barbara, California, claim that a disturbance of the acid base balance, as occurs with heavy protein and other diets, may lead to arteriosclerosis. Certain foods—meat, fish, eggs, cereals and bread—have an acid ash, and render the urine intensely acid. Other foods—vegetables, fruit and milk—leave an alkaline ash. They fed three groups of rabbits on diets in which protein predominated. The first group received liver protein, the second group oat protein, and the third group vegetable protein (soya bean). The protein fed to the first two groups had an acid ash, and that to the last group an alkaline ash. The carbon dioxide content of the serum in the last group (soya bean) was on an average twice as high (59%) as against 30% by volume in control animals. Arteriosclerosis was pronounced in the first two groups, and was practically absent from the third (vegetable protein) group.

It is scarcely necessary to add that these theories, those concerned with altered fat metabolism, diet and disturbed acid base balance, as advanced for the causation of arteriosclerosis, are largely speculative and are not as yet accepted as proven.

Hypertension.

Hypertension as an ætiological factor in the arteriosclerosis of diabetes does not exist. If present, some complication is generally found to account for it. In uncomplicated diabetes the blood pressure is usually within normal limits.

Clinical Manifestations and Practical Applications.

Arteriosclerosis in diabetes involves the arteries of the legs and heart rather than those of the brain. The involvement of the vessels of the legs has already been mentioned and the usefulness of X ray examinations has been made manifest.

Importance of Diet.

In diabetes, when the disease is of long standing or severe, and treatment has been neglected, a high incidence of acidosis and lipæmia occurs. As already shown, arteriosclerosis in such cases is not uncommon. Dietary measures to control these factors are important.

Sansum had this in mind in his advocacy of high carbohydrate diets. He states:

On high carbohydrate diets, with the aid of insulin, patients are entirely freed from the slightest traces of the acetone type of acidosis.

Joslin is of the same opinion:

Insulin helps marvellously in allowing our diabetics to utilize 100 grammes of carbohydrate daily, and thus keep the fat relatively low in the diet, but far lower in the blood, and presumably still lower in the tissues.

Insulin, as everyone knows, provides for complete oxidation of carbohydrate, and this favours the complete oxidation of fat with avoidance of acidosis and lipæmia.

Arteriosclerotic Heart Disease.

Arteriosclerotic heart disease in diabetes is now important. This can be appreciated when it is realized that statistics show that 60% of all cases of diabetes occur in persons over forty years of age and that fifty years of age is the most frequent time of onset. In addition the special factors that have been considered, such as diet, neglect of treatment *et cetera*, in the causation of diabetic arteriosclerosis, must also operate.

Joslin found at autopsy in 40 cases of diabetes in persons over 40 years of age, coronary sclerosis in 24 and sclerosis in the myocardium in 27. Wilder obtained similar results, and especially noted advanced coronary disease in persons who had gangrene of the extremities. The lesson here for the surgeon, and more especially the anæsthetist, is obvious.

The number of references in the literature to coronary sclerosis and *angina pectoris* associated with diabetes is increasing. The danger of hypoglycæmia in these cases is also becoming more apparent. As is well known, the blood sugar figures and the renal threshold figure are often high in arteriosclerosis, many authorities being of opinion that the sclerotic heart and other tissues, in order to be properly nourished, require a level of sugar in the blood that is higher than normal.

Joslin refers to a man who was found dead a few days after he reduced his blood sugar with insulin and diet. He states:

A sudden lowering of the blood sugar level, even to values still above normal, might work disastrously to a heart accustomed for years to work on a much higher blood sugar plane. Therefore, with diabetic patients having *angina pectoris* be extremely cautious.

A. E. Parsonnet and A. S. Hyman, writing in April, 1931, state:

The middle-aged, hyperglycæmic diabetic who is suddenly thrown into the throes of an acute coronary attack following an injection of insulin, is by now a not unfamiliar incident.

They state that Banting in conversation with them stressed the fact that diabetes today is a much over-treated disease. They quote seven patients who had severe coronary seizures following an initial first day dose of insulin. An interference with the glycogen metabolism of the heart is postulated, and they (the authors) avoid giving insulin to diabetics with known cardio-vascular disease until dietary measures have proved insufficient.

An interesting case in point occurred at Sydney Hospital.

C.S., a female, aged fifty-four, was admitted under my care on October 8, 1928, with diabetes and arteriosclerosis. Her weight was 50·4 kilograms (eight stone), her systolic blood pressure was 190 and her diastolic pressure 120 millimetres of mercury. She was placed on a basal diet of 1,200 calories with five units of insulin three times a day, but left the hospital a week later, as she objected to insulin injections.

She was readmitted under the care of Dr. Stokes in February, 1929. A diet of 1,500 calories and insulin injections were given. Attacks of heart block (pulse rate 25) ensued. In hospital the highest fasting blood sugar was 180 milligrammes *per centum*. When the urine was sugar-free, the heart block attacks became more frequent. After leaving the hospital on a diet of 1,650 calories the heart block attacks persisted. She left off insulin and the attacks practically ceased.

I am indebted to Dr. Stokes for his observations.

We considered this patient had an arteriosclerotic heart that probably was not able to get enough sugar to function normally, when the blood sugar was lowered by insulin.

Another patient, admitted under my care in Sydney Hospital, is of interest on account of X ray evidence of calcification of the vessels of the extremities.

S.M., a female, aged seventy years, was admitted on January 3, 1931. A history existed of diabetic symptoms for one year, with recent development of a patch of gangrene (about the size of half-crown) on the left heel. This area became septic and commenced to spread. X ray examination revealed calcification of the vessels of the leg.

The film was of great use in convincing the patient, and more particularly the relatives, of the necessity for amputation at the knee joint. This procedure was carried out on January 29, 1931, with spinal anæsthesia. The flaps sloughed, and the patient died from sepsis and exhaustion some weeks later.

ACKNOWLEDGEMENTS.

In conclusion, I would like to express my thanks to the members of the honorary medical staff of Sydney Hospital for permission to use the case records of diabetics admitted to the medical wards.

TYPHOID CARRIERS: OBSERVATIONS ON THEIR INVESTIGATION AND TREATMENT.

By A. R. Southwood, M.D., M.S.,
Honorary Physician, The Adelaide Hospital,
E. B. Thomas, M.B., B.S.,
Eudunda, South Australia,
AND
R. B. Knight,
University of Adelaide.

The following observations are based on a small epidemic of typhoid fever which occurred between February, 1928, and February, 1931, in the vicinity of Eudunda, a country district about 70 miles north-east of Adelaide. A brief summary is given of the cases and the detection, investigation and treatment of a carrier are described.

Patients suffering from typhoid fever are found during the illness to have *Bacillus typhosus* in the fæces, and often in the urine. During convalescence it is usual for this abnormal state to clear up. A small proportion of patients, probably about 5%, continue for months or years to pass typhoid bacilli with the fæces. These are fæcal carriers. The individual may appear in perfect health, or he may suffer from mild attacks of abdominal discomfort. It has long been recognized that the gall bladder is a favourite habitat for the typhoid organism, and a quiet cholecystitis is the common pathological condition in these typhoid carriers. It is less usual for carriers to pass bacilli in the urine.

Although the typhoid carrier may apparently suffer no ill effects—he may be on perfectly good terms with his germs—he is a great potential danger to the surrounding community. It is to the influence of human typhoid carriers, rather than to persisting soil or water infection, that the endemic occurrence of typhoid fever in certain localities is to be attributed.

Case Reports (E. B. Thomas).

Case I.—P.S., a boy of thirteen years, was taken ill in February, 1928. It was a serious and lengthy illness complicated by several severe hæmorrhages, and convalescence was slow. The patient made a good recovery. The Widal test gave a positive result.

Case II.—P.J.S., the father of P.S., developed typhoid symptoms shortly afterwards. The result of the Widal test was positive and the patient made a good recovery. His wife and the other son were not affected. This family lived in Eudunda township. The source of infection could not be traced.

Case III.—A.M., a man of forty-five years, living at Sutherlands, a village nine miles east of Eudunda, was reported to be ill in April, 1928. He was a farmer and kept a small dairy. The onset of the illness was atypical and a tentative diagnosis of influenza was made. Pyrexia, headache and pains in the limbs occurred, but there were no localizing signs and no rash appeared. In June the patient was admitted to the Adelaide Hospital under the care of Dr. C. T. Ch. de Crespigny. The condition was diagnosed as atypical typhoid fever. The Widal test gave a positive result. The fæces and urine contained typhoid bacilli. In August, while still in hospital, the patient developed signs of cholecystitis. Bile obtained by the duodenal tube gave a culture of typhoid bacilli. The gall bladder inflammation gradually subsided under medical treatment. Subsequent examinations of fæces, urine and bile revealed no typhoid organisms, and the patient

returned home from hospital at the end of September. No definite source of infection was found at the time to account for A.M.'s disease. The family lived close to J.S., who was later found to be a carrier.

CASE IV.—A.P., a girl of eleven years, was reported on June 20, 1928. She had been living at Sutherlands till May 20, and probably derived the infection from there. It is possible that her family obtained milk from A.M.'s dairy.

No further cases were reported in the district in 1928.

CASE V.—G.F., fifty years of age, a labourer in a railway repair gang, was taken ill in April, 1929. J.S., who was later found to be a typhoid carrier, worked in the same gang, and was probably the source of infection. G.F. made a good recovery.

CASE VI.—A nurse at the Eudunda Hospital was reported to be ill in May, 1929. She had nursed G.F. while he was in the hospital, and undoubtedly contracted the disease from him. She was dangerously ill for several weeks, but eventually recovered.

Case VII.—Mrs. M., the wife of A.M. (Case III), became ill in October, 1929. Her husband had been living at home since leaving the Adelaide Hospital in September, 1928. He was suspected of being a carrier, and was sent to the Adelaide Hospital for investigation. Fæces and urine specimens showed no typhoid organisms. The duodenal tube was passed, but no bile was obtained; the duodenal washings showed no typhoid bacilli on culture. The Widal test gave a positive result. After three weeks A.M. was discharged from the hospital. No evidence had been found to convict him of being a carrier at that time.

CASE VIII.—H.C.C., a man at Sutherlands, was seen in March, 1930. The symptoms suggested typhoid fever. There was at first no response to the Widal test, but in the third week a very slight reaction with paratyphoid bacilli was obtained. The patient recovered.

CASE IX.—A.B., a boy of thirteen years, living fourteen miles north of Eudunda, was reported as suffering from typhoid fever in May, 1930. His illness was severe, but he recovered. The source of infection was uncertain.

Another lull occurred. No further cases were reported till the end of 1930. These long intervals between cases made the epidemic very difficult to understand. The remaining seven patients were taken ill within a comparatively short time of one another.

CASE X.—M.F.B., a man of twenty-eight years, of the railway gang in which J.S. (the carrier later detected) worked, was taken ill in November, 1930. Clinically the illness was typically typhoid, but there was no response to the Widal test on two occasions. Pneumonia supervened early in the illness and the patient died. Probably the condition was pneumotyphoid.

CASE XI.—M.B., the daughter of M.F.B., was taken ill shortly after her father. The Widal test gave a positive result. The illness was severe, but the patient recovered.

CASE XII.—F.K., a boy of four years, was also taken ill early in November, 1930. The fæces contained typhoid bacilli. He developed middle ear inflammation and was removed to Adelaide for mastoid operation. The illness was critical, but the boy recovered. This boy's father was also a member of the permanent way gang to which J.S. belonged.

CASE XIII.—E.M., a boy of five years, was a playmate of F.K. Typhoid bacilli were found in the fæces. A good recovery was made.

CASE XIV.—G.C.K., father of F.K., was taken ill in December, 1930. He had a very severe illness, but recovered.

Three other children and the wife of G.C.K. were not affected.

CASE XV.—P.L., a young man living seven miles from Sutherlands, was taken ill. The Widal test gave a strongly positive result. The source of infection was uncertain, but it was probably derived from drinking infected dam water. The patient recovered.

CASE XVI.—L.M., the son of A.M., (Case III) was admitted to hospital in January, 1931. The illness was of a severe type. The Widal test gave a strongly positive result. Death occurred in the fifth week from acute dilatation of the stomach.

The subsequent history of A.M. (Case III) is of interest.

This patient died early in February, 1931, after an illness of five days' duration. The symptoms were those of an acute toxæmia. Autopsy revealed a small contracted and adherent gall bladder filled with pus and containing three small stones.

This was apparently the aftermath of the attack of cholecystitis during his typhoid illness in 1928.

DETECTION OF THE CARRIER (E. B. THOMAS).

When the first of the last series of cases was reported it was clear that A.M. (Case III) was not the only carrier in Sutherlands. Samples of urine and fæces were obtained from all persons in Sutherlands who had typhoid fever at some period in their lives. Specimens were obtained from seven persons. At the same time a public meeting of local residents was called; the gravity of the position was explained and the people were advised to undergo inoculation with typhoid and paratyphoid *A* and *B* vaccine. There was a wonderful response and practically the whole local population was inoculated.

The result of the examination was that all the specimens were normal except those of J.S., a man of sixty years, a labourer in the permanent way gang. He had typhoid fever about six years previously. His urine was normal, but the fæces were found to contain typhoid bacilli. L.G.F., M.F.B. and G.C.K. (Cases V, X and XIV) belonged to the same railway gang. J.S., the carrier, was also a member of this gang, so that infection could readily be (and doubtless was) conveyed from him to the others. Patients number XI and number XII were probably infected through their parents, and so, indirectly, J.S., infected them also.

It is noteworthy, however, that, although J.S., the carrier, spent every week-end at home with his family, none of them became infected.

J.S. was persuaded to go into the Adelaide Hospital for treatment.

Treatment of the Carrier.

The patient, J.S., who appeared to be in excellent health, was admitted to the Adelaide Hospital in February, 1931. Typhoid bacilli were found in the fæces on the two occasions when examinations were made. The urine contained no typhoid bacilli. A duodenal tube was passed, a saturated solution of magnesium sulphate was injected by way of the tube into the duodenum, and a sample of bile was withdrawn. An abundant pure growth of *Bacillus typhosus* was obtained on culture. The count was over 55,000,000 bacilli per cubic centimetre of bile, diluted though it was by the magnesium sulphate.

Cholecystectomy was advised and the operation was performed by Sir Henry Newland. The gall bladder was surrounded by dense adhesions; it was enlarged and its walls were thickened. There were also adhesions from the whole of the superior surface of the liver to the diaphragm.

On section of the gall bladder, typhoid bacilli were seen on the surface of the epithelium, but none could be found beneath, although there were areas of round cell accumulation indicating a subacute inflammatory reaction.

Following the operation on March 14 bacilli were again found in the fæces, but not in the urine. On March 23 a sample of blood was taken and no agglutination was obtained either with a living emulsion (O-agglutinins) or with formalinized emulsion (H-agglutinins) of *Bacillus typhosus*. On March 23 neither urine nor fæces gave any response to tests for typhoid bacilli. Tests were repeated on April 2 and April 9 without result.

On April 16 the duodenal tube was passed again and a sample of bile was withdrawn. There was no growth on attempted culture.

If bacilli had persisted in the bile ducts (cholangitis) it was expected that this second examination of bile would reveal their presence. The failure to detect any abnormality made it highly improbable that the patient was still a carrier, and he was therefore discharged as free from infection. It is proposed to repeat examinations of urine, fæces and bile at intervals during the next year.

Conclusions and Comments (A. R. Southwood).

1. *The Importance of the Human Carrier as a Vector in Typhoid.*—Adequate supervision of the convalescent patient is essential. It is usual to insist on three normal findings in the bacteriological examination of fæces and urine; in most cases this precaution suffices, but intermittent excretion of organisms may allow some carriers to escape detection. The problem is a difficult one. When a typhoid outbreak occurs, the public health authority concerned must institute a vigorous search for human carriers. Contacts giving history of typhoid infection, especially if the illness is at all recent, must be regarded with suspicion, and induced to submit to investigation. It is the duty of the private practitioner to cooperate actively in the search. In the small outbreak under review Dr. E. B. Thomas was successful in detecting the carrier and in persuading him to submit to treatment.

2. *Difficulties in a Small Epidemic Spread Over a Long Period and with Scattered Cases.*—The source of sporadic infections is generally obscure. Careful inquiry for possible carriers and examination of specimens of fæces and urine are essential, tedious and lengthy processes through they be. Enthusiasm, keen observation, diplomacy and tact are necessary in handling the situation.

3. *The Manifestations of Typhoid Fever.*—Typhoid fever is protean in its manifestations. Mild cases of persistent pyrexia or of enteritis, and obscure forms of bronchitis or pneumonia should be investigated carefully and bacteriological examination made of all suspected cases.

4. *The Necessity for Protective Inoculation.*—In hospitals where typhoid patients are admitted, compulsory inoculation of nurses should be observed. In Case VI of the present series, infection was transferred from patient to nurse. The value of protective inoculation was stressed by Osler many years before it met with general acceptance by the medical profession. Striking proof is provided by the comparison of the low incidence of typhoid infections in the Great War with the very high incidence in the American Civil, the Franco-Prussian, the Spanish-American and the South African wars.

5. *Vagaries of the Carrier.*—The passage of infective organisms from the carrier is generally intermittent. There may be intervals of several months when no bacilli can be detected. Even the fact of three tests of the urine and fæces of the convalescent typhoid patient giving normal results is not conclusive evidence that he harbours no organisms. The difficulty might be met by repeated examinations of excreta at intervals of two months during the year following the illness. A.M. (Case III) was apparently infection-free after his illness in 1928, and further investigation in 1929 gave normal findings. The illness of his wife, however, and his own death from suppurative cholecystitis suggest the probability of his being a chronic carrier.

6. *The Value of the Duodenal Tube in Detecting Infection of the Biliary Tract.*—Although it is generally accepted as a fact that the fæcal carrier harbours the organisms in the gall bladder, it is desirable in any case where cholecystectomy is contemplated to ascertain definitely whether typhoid bacilli are present in the bile. After the operation, in addition to the usual bacteriological investigation of fæces and urine, specimens of bile obtained by duodenal intubation should be examined. In this way any persisting infection of the biliary tract, such as cholangitis, may be detected.

7. *Efficacy of Cholecystectomy in the Treatment of Chronic Carriers.*—It is authoritatively stated that cholecystectomy will clear up the infective focus in about 80% of chronic typhoid carriers. Its value is comparable with that of tonsillectomy in ridding the chronic diphtheria carrier of his focus. For patients convalescing from an attack of typhoid fever, medical treatment with hexamine and repeated drainage of the gall bladder by duodenal intubation and magnesium sulphate injection is advised while the carrier state persists.

8. *The Doctor and Preventive Medicine.*—Every medical practitioner should cultivate the preventive outlook. The occurrence of a case of typhoid fever requires not only that the patient be treated, investigated and kept under proper observation till infection-free, but also that carriers be sought for. The preventive aspect of medicine is so vital that it should colour the whole course of the student's university and hospital work. Many of the duties involved in the conduct of preventive measures may be irksome, but, as the great Osler said, in preventive medicine nothing necessary is irksome.

Acknowledgement.

Throughout the recurrence of the cases the Local Board of Health and its officers were diligent in their efforts to trace the disease to its source and in their endeavours to prevent any further cases occurring. Assistance was rendered by the Central Board of Health by the services of one of its officers being made available in making detailed inquiries into the cases.

Appreciation must be expressed of the work of the staff of the South Australian Government Laboratory, under the direction of Dr. L. B. Bull, in carrying out the bacteriological investigations.

AFFECTIONS OF PERIPHERAL NERVES.[1]

By A. W. Campbell, M.D.,
Sydney.

The subject chosen for our deliberation this evening, "Affections of Peripheral Nerves," obviously covers a field so wide that in opening this discussion I can make only a cursory survey and selectively indicate some topics which seem to be worthy.

Such affections are customarily and conveniently divided into three groups, namely, paralytic, neuritic and neuralgic.

Paralytic Affections.

Paralytic affections include all those paralyses consequent upon lesions of individual nerves or plexuses of nerves. Such lesions are produced much more by trauma than by disease and were most abundantly exemplified among the wounded in the Great War; indeed as a result of this tragedy we saw hundreds of cases for every one case we had previously seen, and so were provided with such an abundance of new data that all chapters on the subject had to be remodelled and some entirely rewritten.

One early and important lesson was that the effect of a bullet upon a peripheral nerve was very different from that of a simple experimental or accidental cut. We soon realized that every time a nerve was injured the reaction was peculiar to that case and was so not only because the various nerves were found to have differing individualities, but because the condition of the wound and the destructive changes in the surrounding tissues were different on each occasion. Moreover, surgeons can testify that when they came to measures of repair, they found that while the cicatrices of nerves which had been injured or sutured or resected or freed from adhesions, might bear a superficial resemblance to one another, the resulting clinical course would present infinite variety and often confound expectation.

I have mentioned that various nerves were found to have differing individualities, and may add that this was no more strikingly shown than when we had to deal with painful paralytic lesions. I refer to what is termed causalgia. For instance, in the case of wounds of the sciatic nerve our experience was such that it came to be laid down as an almost invariable rule that there would be causalgia only when fibres pertaining to the internal popliteal nerve were involved. And correspondingly in the arm, painful lesions were mainly confined to the median nerve. From this it is not to be gathered that other nerves were wholly immune, but in them causalgia was very uncommon, and if pain did arise, it was of other character and usually referable to some complication, such as fracture.

The pathogeny of causalgia is for your consideration. Sundry hypotheses have been put forth, but not one, I think, has been found generally acceptable.

Further, in regard to analogies, the way in which the musculo-spiral nerve in the arm resembled the external popliteal or peroneal in the leg was remarkable. On account of the exposed position of both, injuries were very frequent; in both it was most unusual to see either painful states or serious sensory or trophic disturbances, and both lent themselves more willingly than other nerves to processes of repair.

Then it was a novel experience to meet with so many lesions of nerves associated with lesions of blood vessels. The appearances presented by these mixed lesions had been occasionally observed prior to the war and had been attributed to complete interruption or serious lesion of mixed peripheral nerves, but the war proved that they arose only when there was an arterial lesion added to the nerve lesion. The appearances in question were of a trophic nature. They included glossy skin, deformed nails, œdema of subcutaneous tissues illustrated by the well known "succulent hand," extraordinary induration of muscles, and unusual swelling of joints. These changes, in themselves sufficiently striking, were attended by anomalous sensory phenomena: thus anæsthesia which had a segmental rather than a peripheral nerve distribution, and pain, different from that of causalgia in being less severe, less localized and less tractable.

Lastly, I may say that a number of other matters bearing on paralyses of peripheral nerves still remain which I must leave untouched, for instance, the various clinical syndromes as they affect lesions of nerves, both in particular and in general, which include the syndromes of complete interruption, of irritation, of dissociation (partial lesions), of compression (lesion around more than within the nerve) and of regeneration and the whole process of recovery and treatment.

Neuritic Affections.

Neuritic affections of peripheral nerves are divisible into two main classes, local, that is, confined to one nerve trunk, and multiple, of which alcoholic neuritis is an example.

Concerning local neuritis I may first comment on the frequency, freedom and falsity with which this title is tacked on to sundry pains. Pain alone is not adequate to the diagnosis of neuritis. I take it that additional postulates are paræsthesia (tingling, formication *et cetera*) and possibly some sensory loss, both, like the pain, in the distribution of the peripheral nerve affected; also pain in the nerve trunk, if this be palpable, and a degree of muscular paralysis or wasting, if the affected nerve be motor or mixed. Should these postulates be not forthcoming and pain alone exist, the condition must be termed neuralgia. More often perhaps the correct diagnosis might be myalgia, arthritis, fibrositis or other inflammatory process having nothing to do with the peripheral nerve.

Next it may be indicated that a pure neuritis of one peripheral nerve is uncommon. An isolated affection of a peripheral nerve, the outcome of pressure or some such local lesion, is not, strictly speak-

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 27, 1931.

ing, a neuritis; it is a peripheral palsy. I think of crutch palsy, conditions associable with cervical ribs and with *spondylitis deformans*, pressure or contusion lesions of the musculo-spiral or peroneal nerves, certain craft palsies, and so on. Here, by the way, I may mention a curious instance of craft palsy in the case of a woman whose daily task it was to fill boxes with butter. Constantly repeated pressure upon the wrist in sliding a board over the top of each box in the final manœuvre had induced paralysis of the ulnar nerve where it enters the hand.

As an example of a pure local neuritis we may take leprous neuritis, where the lepra bacillus multiplies in the interstitial tissues of a nerve, also some cases of syphilitic neuritis, and that neuritis which will occasionally follow infectious disease. Thus I have seen neuritis of the musculo-spiral nerve after both enteric and puerperal fever. But concerning neuritis of infective origin, it is singular that it is not more frequent, especially if what some experimenters tell us be true, namely, that infective processes travel from periphery to spinal cord by way of peripheral nerves. One would imagine that organisms would be often taking this journey and that we would often see an isolated ascending neuritis, which is not the case.

There are many kinds of multiple neuritis or polyneuritis, resembling each other more or less in clinical character, but differing in causation, and accordingly, until our history sheet is complete, not easy of exact diagnosis. By way of illustrating this, let me say that I happened to be working in Lancashire during a classical outbreak of multiple neuritis caused by arsenical poisoning. In the beginning the sudden, widespread and apparently epidemic nature of the affection made observers hesitant in diagnosis, and not until several moons had passed was all made clear by the discovery of beer contaminated with arsenic.

Mention of beer leads on to alcoholic neuritis and one's first remark is by way of a query. Why is the ratio between the cases of alcoholic neuritis we see and the quantity of alcohol consumed so disproportionate? In addition to the alcohol is there any other determining or precipitating factor?

Another not fully recognized point concerning alcoholic neuritis and one perhaps applicable to most other forms of multiple neuritis, is that degenerative changes occur not only in the peripheral nerves, but throughout the central nervous system. I may add that if in any case of toxic neuritis you wish to obtain good histological pictures of the changes, examine the external popliteal, musculo-spiral and vagus nerves. Lead neuritis I shall pass by with this one remark. We are given to believe that neuritis, and particularly optic neuritis, ascribed to plumbism is of frequent occurrence in Queensland. Is it not possible that the same affection is more common than we suppose in New South Wales?

Next we have a well recognized form of acute and often febrile multiple neuritis which, though speculatively ascribed to infection or excessive cold and damp, or autotoxæmia, is actually of obscure origin. We saw many such cases during the Great War; we also see them here, and as their features are well known, I need say no more about them.

Less commonly we have multiple neuritis, which is definitely to be coupled with infective and biochemical and other disturbances. I need only mention those occasionally following typhoid and paratyphoid fever and septicæmia and the polyneuritis of diabetes and beri-beri, and others associated with tuberculous and malignant cachexias, and senile polyneuritis and neuritic forms of encephalitis.

Lastly, there are cases of progressive multiple neuritis, with or without hypertrophy of nerve trunks, of which the dominating clinical feature is the extreme muscular weakness and wasting, most distressing to look upon and fatally progressive in course. The ætiology of these conditions is obscure, and happily they are rare.

Neuralgic Affections.

Neuralgic affections may be considered according to anatomical distribution.

Sciatica is perhaps the most typical example of a painful affection of a peripheral nerve and it is one about which much might be said. Indeed, as some of us know, at the last international meeting of neurologists held in Paris sciatica was taken as the subject for discussion and was looked at with French thoroughness from every angle. The opener of the discussion, after referring to numerous pseudosciatic affections, divided true sciatica into five groups: (i) funicular, characterized by lumbago; (ii) plexular, characterized by sacro-iliac pain; (iii) truncular, which is the common and to us well known form; (iv) muscular, in which the pain is confined wholly to hamstring muscles; and (v) cellular, characterized by the appearance of cellular or shotty nodules in subcutaneous tissues and other parts. Without denying the existence of all these forms, I think we will agree that the so-called truncular form is for us the form of most importance and perhaps that which specially yields to treatment.

And in the matter of treatment we all have our favoured measures, which may be medication, rest, electricity or flooding the nerve with normal saline solution. Personally I like the last measure and have notes of many cases in which the result has been most gratifying.

Brachial neuralgia in the arm or shoulder corresponds with sciatica in the leg, but in my experience is much less tractable. It would seem to be mainly, though not wholly, an affection of women, and it is a question whether we are to agree with those who state that the subjects are invariably tinctured with neurosis. For me the statement does not carry full conviction.

In the diagnosis of brachial neuralgia there is much scope for error. X ray examination in one of my supposed cases revealed early tuberculosis of

the shoulder joint, and in many some other form of arthritis has been disclosed. I have also seen the pain of brachial neuralgia exactly simulated and undoubtedly conditioned by spondylitis affecting cervical vertebræ. And over and over again a painful bursa or a fibrositis or a limited and localizable myalgia has been the correct diagnosis. Plainly, therefore, we must be circumspect before we commit ourselves.

Occipital neuralgia, pure and uncomplicated, is a more clearly cut affection, its distribution along the occipital nerves and its characteristic tender points make diagnosis easy.

Turning to the association between neuralgic affections and *spondylitis deformans*, I am sure that any having dealings with applicants for pension among returned soldiers will be satisfied as to the frequency of this association. In its wisdom the Department of Repatriation makes free use of its X ray apparatus and it is surprising what a number of men, mostly middle aged, complaining of aches and pains in trunk and limbs, are discovered to have *spondylitis deformans* as the almost certain cause of their trouble. The radiologist is apt to ascribe these pains to pressure of bone upon emerging nerves. Perhaps it is more correct to assume pressure of inflammatory changes in contiguous soft parts, changes which must accompany the bony deformity and which the X rays cannot reveal.

Probably that very common and to the patient very alarming condition, when it chances to strike over the heart, intercostal neuralgia, is sometimes referable to spondylitis and the same might be said of many lumbagos.

Finally we come to trigeminal neuralgia (*tic douloureux*), than which there is no more painful human affliction. Observation of this disease teaches several lessons. First, in the matter of diagnosis it is well to remember it is a disease of advancing years. When a person under forty-five comes complaining of neuralgia, my practice is immediately to set in train investigation for diseased sinuses or other cause of facial pain. Secondly, be chary of certain nervous old people of both sexes who lay claim to the possession of *tic douloureux*. They make much of their case, but close observation will prove that they suffer differently from the genuine victim; their pain will not come in those extraordinary spasms and they will lack other accompaniments which stamp true *tic douloureux*. They have been given the ugly name "neurotic neuralgics," and the whole gamut of treatment by drug, injection, neurectomy and gasserectomy will certainly fail to bring them relief. And, thirdly, in regard to treatment of the true affection, a personal experience of close on one hundred and fifty cases has convinced me that injections of alcohol, either through peripheral foramina or into the gasserian ganglion itself, according to circumstance, provides an excellent means of treatment. It is certainly as effective as a peripheral neurectomy or an avulsion, and should be tried before radical intracranial operation is undertaken.

AFFECTIONS OF THE PERIPHERAL NERVES.[1]

By J. C. B. Allen, M.B., Ch.M. (Sydney), F.R.C.S. (England),
Sydney.

Speaking broadly, affections of peripheral nerves which come under the notice of the orthopædic surgeon are those resulting either from an anatomical or a physiological block. Of this group the traumatic affections undoubtedly make up the largest proportion.

The peripheral nerve is comprised of neurones subserving either sensory or motor functions, usually mixed, but sometimes, as in the case of the digital nerve, one function only is carried out.

Trauma brings about a condition of either partial or complete interruption of conductivity. On the one hand there is a loss of sensation and on the other loss of muscular activity results. Varying grades of disability and of deformity occur, and it is these which bring the conditions under our notice.

General Principles.

Certain general principles can be laid down for the treatment of paralytic conditions. First, it is essential to repair damage which has occurred in the nerve, to free it from the external influences of pressure and traction by adhesions. Secondly, there is the necessity of keeping the paralysed muscles at rest and in a position of relaxation. This point must be stressed. Unless the affected muscle is put in its optimum position for recovery, degeneration from stretching will occur; and if the nerve supply is subsequently restored, the muscle is unable to carry out its allotted function and its usefulness has ceased to exist. Intimately associated with this question of relaxing paralysed muscles is the necessity of placing joints in their position of rest. Nothing will hasten the stiffening of a joint to such an extent as its fixation in a position of strain. Wherever possible this principle should be observed. It is sometimes, however, necessary to sacrifice the position of the joint to adhere to the dictates of the paralysed muscle, for example, in a musculo-spiral palsy the wrist must be maintained in dorsiflexion, whereas the wrist is relaxed in 5° palmar flexion.

Finally, we come to the question of physiotherapy. This branch of treatment is a further essential, and it should progress along definite lines. First, galvanism should be applied to the affected muscles in order to maintain artificially as far as is possible their tone and vitality. When signs of recovery appear in the nerve, galvanism should be replaced by some type of faradic current which, it is needless to point out, stimulates the muscles only through their nerve supply. Lastly, voluntary movements which not uncommonly appear before response occurs with the Bristow battery, should be encouraged from the outset. To repeat the old adage, "an ounce of voluntary movement is worth a

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on August 27, 1931.

ton of massage and electricity," and as soon as it is possible, active movements must be encouraged in every possible way by those in control of the patient. The interest of the patient must be stimulated so that he is prepared to give as much assistance as possible in the treatment of his own condition.

At the present time there is a tendency to decry mechanical devices in the treatment of these conditions. This, I do not think, is reasonable, as a well devised machine which, however, depends for its working on the effort of the patient, adds greatly to the variety of treatment and absence of monotony. A bicycle, for example. A man in middle life will often get considerable satisfaction from being able to tell his friends of like age that he has ridden three miles, or whatever the distance may be, and he will increase his effort to break his previous record. These machines need not necessarily be accurate, and even though he may only ride a quarter of a mile, the exaggeration is justifiable.

Finally, with regard to treatment, we come to those cases in which recovery of the nerve has failed to take place. Various disabilities and deformities are the result, and in many cases considerable ingenuity is required to alleviate these conditions. This subject is naturally one of considerable magnitude, and apart from mentioning a few possibilities when referring to specific instances, I do not propose to deal with it.

Individual Nerves.

The Facial Nerve.

Passing to the consideration of individual nerves, let us take first of all the facial nerve. The conditions chiefly seen are facial paralyses following trauma to the nerve in the course of a radical mastoid operation, and secondly, a Bell's palsy. It would appear needless to enumerate the unpleasant symptoms and signs of the condition, beyond remarking on their distressing effect both on the patient and on his associates. Cosmetically the picture can only be described as horrible. Epiphora, dribbling of saliva, and the collection of food in the cheeks give rise to considerable inconvenience, and the inability to close the eye leads not infrequently to serious complications.

Treatment should be commenced as soon as the condition is recognized. To obviate muscle stretching and subsequent contractures the angle of the mouth should be elevated by means of a splint, the other end of which hooks around the ear; the ear piece of the ordinary spectacles slightly modified is the most satisfactory type to use. Gentle massage and electricity together with efforts at voluntary movement should be commenced at the same time. The majority of these paralyses clear up satisfactorily, leaving no disability or disfigurement. There is, however, a percentage both of the traumatic group and of the Bell's palsies in which recovery does not take place, and it is this group which, I think, deserve a deeper consideration than is very often given to them. Nerve anastomoses with the seventh nerve give probably some of the most satisfactory results of this type of operation, and consequently I propose to go a little more fully into the types of operation which have been suggested and carried out.

First, it is necessary to select cases carefully, and in this connexion it is obviously useless to carry out any type of anastomosis when there is absence of response on the part of the facial muscles to any type of electrical stimulation. It is therefore necessary to test carefully the electrical reactions of the muscles in question. If a response is obtained, the possibility of improvement or recovery justifies operative interference. Recovery after operation is in three stages. In the first there is an increase in tone. It is suggested that this alone is sufficient reason to justify operation, as recovery to a large extent occurs in the condition of the eye. Epiphora is less, and the eversion of the lower lid is diminished. The second stage is that of recovery of voluntary movement. Ballance is of the opinion that this is likely to follow. He thinks that the third stage, which is the return of expressional movements, is unlikely to occur except in children.

As regards the time at which operative interference should be undertaken, a period of six months' adequate treatment is always advisable. If at the end of this time no recovery is manifested, operation should be carried out.

Concerning the operation itself, various nerves have been suggested and experimented with for anastomosis. Following the work of Ballance, end-to-end union would appear to be the procedure of choice, and according to his conclusions after a large series of investigations, the glosso-pharyngeal is the ideal nerve to select for the grafting. He quotes a case in which this nerve was anastomosed to the facial in paralysis following a radical mastoid operation sixteen years previously. Recovery commenced at the end of four months and progressed to the completed second stage.

Amongst the other nerves which have given fairly satisfactory results, are the hypoglossal, the division of which leaves practically no disability in spite of the fact that the homolateral side of the tongue atrophies. Recovery usually follows to the completed second stage.

The spinal accessory nerve has also been used, but was given up many years ago by Ballance owing to the fact that dissociated movements rarely, if ever, occurred, movements of the face being accompanied by a shoulder movement. At the same time, weakness of the shoulder led to some disability in those who were carrying out manual labour.

Passing now to those cases in which complete degeneration has occurred in the facial muscles, some type of reconstruction operation is considered to bring about a certain amount of relief. Various operations have been suggested, and it may be of interest to mention some of them in passing.

The object in each instance is to elevate the angle of the mouth and to increase the tension of

and to support the lower eyelid. Grafts from the temporal muscle into the *orbicularis oculi*, facial grafts from the temporal fascia to the angle of the mouth, together with excisions of the skin and fat over the malar bone with subsequent suturing under tension and resection of the whole thickness of the cheek in the region of the naso-labial fold beyond the angle of the mouth, are recommended by Porter. Lodge recommends the use of a graft of the *fascia lata*, inserted beyond the outer canthus of the eye, looping around the internal palpebral ligament and the *orbicularis oris*, to emerge again at its point of entrance. The free ends are drawn taught and adjusted according to requirements and then made fast to the fascia over the temporal muscle. These operations have the great advantage of an immediate effect in lessening the unpleasant symptoms of the condition.

The Brachial Plexus.

In the brachial plexus group come the majority of the birth palsies, that most commonly seen being the Erb or upper plexus type. This condition is attributed to stretching and rupture of the fibres of the fifth and at times the sixth cervical nerve by a wide separation of the head and shoulder at the time of birth. This leads to a well recognized and not uncommon condition in which the muscles affected are the external rotators of the shoulder, the abductors, the biceps and not uncommonly the extensors of the thumb and the *supinator longus*. The arm is in the familiar position known as the policeman's tip.

Treatment consists in splinting the arm in abduction, external rotation, flexion at the elbow and full supination. Recovery in the majority of cases takes place, but occasionally, in a neglected case particularly, the position at the shoulder of internal rotation and of pronation in the forearm persists. Considerable relief in these cases can be obtained by dividing contracted muscles, such as the subscapularis and the pectorals, in the region of the shoulder and the *pronator radii teres* in the forearm, and allowing the contracted positions to be corrected.

The lower plexus lesion is much less uncommon and affects the first thoracic and eight cervical branches to the plexus. It occurs occasionally as a birth palsy in breech deliveries with an extended arm. A further occasional causative factor is grasping something while falling, the arm being wrenched from the trunk. In this lesion anæsthesia occurs over the ulnar distribution in the forearm and hand, and the intrinsic muscles of the hand are paralysed, including the muscles of the thenar and hypothenar groups. Numerous cases of both this and of the previous type have been reported from bullet wounds in the region of the neck. Little hope of recovery can be held out in these cases due to trauma. Attempts to suture ruptured nerves will almost invariably end in failure owing to the difficulty in isolating the severed ends, and for the further reason that very often the lesion appears to be an avulsion almost at its emergence from the cord.

Complete Plexus Injury.

Complete plexus injury is uncommon, but occurs occasionally with fractures of the clavicle. The arm becomes completely useless; all muscle groups are affected. Attempts at open operations with the object of repairing the damage are hopeless. It is impossible to distinguish one branch or even part of the plexus from another, and it has been my doubtful pleasure to disarticulate the useless upper limb at the shoulder joint at the request of the patient in one of these cases.

Musculo-Spiral Nerve.

The musculo-spiral nerve is said most commonly to be that associated with fractures, and a glimpse at its anatomy in relation to the humerus will serve to explain the reason for this. It lies during its course in the middle third of the arm in close relation to the bone, and in view of its spiral course it is particularly liable to trauma resulting from displacement of the fragments. Paralysis here is of two types, primary and secondary. The primary occurs at the time of the fracture, either by direct injury or secondary to the bone lesion. The secondary group occurs during the healing of the fracture and results from callus inclusion; the callus by pressure impairs the function of the nerve, even up to total paralysis. It is further suggested that stretching over the mass of callus may result in a loss of function. The symptoms are, of course, a paralysis of the extensor muscles of the wrist and fingers, either partial or complete. Sensory loss occurs on the dorsum of the hand between the thumb and the second metacarpal, the wrist being in a position of palmar flexion. The Saturday night paralysis that one remembers hearing so much about in one's student days, resulting from the arm hanging over the back of a chair, is now rarely seen. Crutch paralysis, however, due to a similar cause is occasionally met with.

As regards treatment, the general opinion is that 50% of patients with this condition require operation. Apart from open fracture, operation should be delayed and splinting and physio-therapy persisted in for from four to six months. If at the end of this time improvement has not occurred, exploration seems to be indicated. Secondary paralysis, however, never recurs spontaneously and delay in these circumstances serves no useful purpose. Here again suture of the divided ends is the ideal operation when the nerve is divided; when it is included in callus, freeing and adequate protection by muscle is all that is required. In primary cases it is sometimes necessary to resect a portion of the humerus to allow end-to-end apposition of the nerve.

Ulnar Nerve Lesions.

Paralyses of the ulnar nerve associated with trauma are of two distinct types, as in paralysis of the musculo-spiral nerve, with this difference, that it is extremely rare to find an anatomical division of the nerve. Primary paralysis occurs in association with fractures most frequently of the internal

condyle of the humerus, rarely in supracondylar fractures. Platt reports that a consecutive series of 329 cases of fracture in the region of the elbow joint was made up as follows:

Humerus—	
Supracondylar fractures	95
Fractures of the internal condyle	74
Fractures of the external condyle	83
Olecranon	38
Radius, head and neck	39
Total	329

There were nine cases of associated nerve injury. In each instance the affected nerve was the ulnar. Eight accompanied fractures of the internal condyle and one a supracondylar fracture. Spontaneous recovery in these conditions is usual, and in only one case reported by Platt was there a persistence of clinical signs necessitating exploration. The clinical picture is a paralysis of the intrinsic muscles of the hand, and a complete sensory loss over the ulnar distribution. The explanation of the condition, which occurs immediately following the fracture or within the first few weeks, is either actual damage to the nerve by the fractured surface of the bone, or stretching by callus and friction over the callus, brought about by movements at the elbow joint. It is not uncommon to see the nerve abnormally adherent to the surrounding structures and consequently suffering by reason of the joint movements. Joint movement may further bring about in the early days repeated traumata to the nerve by sharp fragments in the region of the fracture. It will thus be seen that while the immediate injury to the nerve is beyond the control of the surgeon, those injuries occurring at the end of a few weeks may reasonably be attributed to the treatment which is being carried out, and signs of ulnar nerve involvement serve to indicate the necessity for rest of the part and the inadvisability of continued mobilization of the elbow. Recovery with a cessation of treatment is the rule.

The second type, that of late ulnar nerve involvement after fractures, is an interesting and long recognized clinical entity which was described originally by Panas in 1878. Since that time many surgeons have given it their attention, and within recent years Platt has made it a special object of study. Platt's conclusions are that the initial injury is usually sustained in early life between the ages of two and ten. It takes the form of a fracture of the external condyle of the humerus splitting obliquely into the elbow joint. Displacement occurs by reason of the pull of the extensor tendons, and union takes place by fibrous tissue. The function of the joint is as a rule fairly satisfactory, *cubitus valgus* tends to occur comparatively early, but causes little disability. There is then a latent period over a varying number of years, even up to thirty, and Platt maintains that in 75% it is not less than ten years. The symptoms of ulnar nerve involvement are insidious and progressive, and commence either as a sensory or motor group. The sensory symptoms are pain in the region of the elbow joint with tingling and numbness in the ulnar distribution. The motor group is manifested by a slowly progressing weakness of the hand with increasing difficulty in carrying out the finer movements. The objective signs are those of an incomplete nerve block which go on to complete motor and sensory paralysis. There is weakness and wasting of the intrinsic hand muscles, a partial sensory loss, and finally a definite anæsthesia. Trophic lesions occur to a greater or less extent. Palpation of the nerve in the post-condylar groove demonstrates definite tenderness and possible thickening of the nerve. *Cubitus valgus*, already referred to, is invariably present, and suggests an overgrowth of the internal condyle. This, however, is readily seen not to be the case; on examination of the elbow joint with X rays the old fracture of the external condyle with no sign of bony union is revealed. Mouchet explains the reason for the ulnar nerve condition by pointing out that the elbow deformity necessitates the nerve taking a longer course and thus being put to an abnormal tension with movements of the elbow joint; this he explains as a friction neuritis. In support of this view it is often noticed that symptoms first make their appearance coincident with a period of overactivity of the elbow joint.

Treatment.

Numerous procedures have been suggested: (i) Simple freeing of the nerve, (ii) gouging out of the post-condyle of groove, (iii) supracondylar osteotomy, and (iv) anterior transposition of the nerve. At the present time anterior transposition of the nerve is the operation of choice. The nerve when exposed is often found to be enlarged, adherent to its sheath and surrounding structures, and the groove is shallow. A new bed is made in front of the internal condyle in the muscles attached thereto. This operation fulfils the necessary requirements, namely, to shorten the course of the nerve and to eliminate tension and friction.

Median Nerve Lesion.

Apart from lacerated wounds in front of the wrist, the median nerve rarely suffers from the effects from trauma. Partial or complete division in this situation, when it does occur, leads to considerable disability. Anæsthesia is present over the median area of the palm and fingers and from the point of view of usefulness the function of the hand is very considerably impaired. All the finer movements are lost with this loss of sensation. The short muscles of the thumb, with the exception of portion of the *adductor policis*, are paralysed. Repair of the nerve should always be attempted. Its isolation and suture in the majority of cases is not difficult, but unfortunately the results are disappointing, regeneration rarely being complete.

It is particularly with the lesion of this nerve that the most distressing condition, known as causalgia, occurs. It occurs following an incomplete division of the nerve, often accompanied by sepsis, and is manifested by extreme pain referred to the

palm of the hand. A similar condition occurs in the sole of the foot following an incomplete lesion of the sciatic nerve. A point of interest in these cases is the remarkable success which has been achieved by periarterial sympathectomy in the relief of pain. This operation has now entirely replaced the older one of division of the posterior nerve roots.

Sensory Nerves of the Fingers.

Lesions of the sensory nerves of the fingers are not at all uncommon in injuries to the fingers and lead to the well-known loss of sensation and wasting of the finger pads. Often they are associated with tendon injuries, the repair of which is never satisfactory apart from repair of the nerve, as a finger that has lost its sensation has lost 75% of its function. Not only that, trophic lesions are prone to occur. Bunnell points out that the repair of these nerves is most satisfactory, as they are not mixed nerves and, being distant from the central nervous system, regenerate rapidly. Repair is possible up to the distal crease of the finger.

Nerves of the Lower Limb.

The nerves of the lower limb are rarely involved by trauma. Gunshot wounds, of course, are responsible for nerve injuries in the lower limb, as they are in other situations, but more rarely. Amongst the few lesions that are seen, is the involvement of the fifth lumbar anterior primary division as it crosses the brim of the pelvis. Compression here occurs during parturition, there being either a large head or some abnormality of the pelvic brim which brings this nerve into more than usual prominence. The resulting symptoms resemble those caused by a division of the external popliteal nerve with the difference that the *tibialis anticus* is usually unaffected and sensation is unimpaired. At the same time a consideration of the history will lead to a correct interpretation of the condition.

The External Popliteal Nerve.

The external popliteal nerve suffers from trauma in that portion of its course where it winds around the neck of the fibula. It is here comparatively superficial and unprotected from injury. Direct violence may affect the nerve alone or may affect it in conjunction with the fibula itself, a fracture resulting and the nerve suffering as a primary injury. The condition is one similar to the previous lesion described, but to reiterate, the *tibialis anticus* is involved and there is a loss of sensation over the area supplied by the nerve. The foot is in a position of *talipes equino-varus* with the *tibialis anticus*, extensors of the toes and the peroneal muscles paralysed.

Treatment consists in splinting the foot in a corrected position and in the application of the requisite physio-therapy to the muscles. Subsequently it is advisable for the patient to wear a boot with a toe lift and a night shoe for a considerable period of time.

The Sciatic Nerve.

The sciatic nerve rarely comes in for injury and, apart from the condition of causalgia following an incomplete division, its lesions come within the province of the physician.

Pain in the Knee after Operation.

Lastly, there is one distressing condition which I should like to mention and that is the pain which not infrequently follows operations on medial cartilages of the knee. This pain is situated in the region of and distal to the scar. In my hands recovery has invariably taken place after resection of the scar and a portion of the infrapatellar branch of the internal saphenous nerve, which has become involved in the scar tissue.

Reports of Cases.

THYREOID DISEASE.

By Geoffrey A. Penington, M.D. (Melbourne),
Honorary Physician to Out-Patients, Melbourne Hospital.

The following cases of thyreoid disease were presented at a meeting of the Victorian Branch of the British Medical Association, held at the Melbourne Hospital on May 20, 1931, and illustrate certain typical and atypical forms.

Myxœdema.

Mrs. R.C., fifty-five years old, attended at the hospital on August 27, 1925, complaining of swelling of the face for two weeks. She thought that she was suffering from erysipelas. She had felt the cold very much during the preceding few months, especially in the winter, and had noticed that for eighteen months speech and movements were becoming slower, she was always wanting to go to sleep, and her hands were getting larger. Her hair had not been falling out, bowel function was normal, and menstruation had ceased ten years previously.

Examination disclosed a classical picture of myxœdema. The face appeared to be swollen, with puffiness of the eyelids, thick lips, broad nostrils, a sallow complexion, and redness over the malar prominences—the so-called "apple blossom" cheeks. The mucous membranes looked thickened in the mouth and nose, and were paler than normal. The skin felt thick and dry, was rough and slightly scaly and remarkably hairless over the trunk and limbs. The ears were swollen; large, soft, gelatinous pads, like fat, were present in the supraclavicular regions. The hands were large and spade-like, the fingers being thick and clumsy. The limbs appeared œdematous, but there was no pitting on pressure. The hair was coarse and dry, and marked *seborrhœa capitis* was present. The speech was slow and the voice husky. Mental processes were slow, the patient being sleepy and undecided in her response to questions.

There was no evidence of any cardio-vascular or renal lesion; the pulse rate was 72 per minute, the systolic blood pressure 140 and the diastolic pressure 105 millimetres of mercury. Her height was 180 centimetres (six feet) and her weight 81 kilograms (twelve stone twelve pounds); the basal metabolic rate was found to be – 42%.

Thyreoid gland was administered, in doses of 0·03 gramme (half a grain) three times a day, and in three days she reported that she felt better than previously. Three days later the dose was increased to 0·06 gramme (one grain) thrice daily, and in twenty-five days she lost 4·5 kilograms (ten pounds) in weight, and had greatly improved. Within two months she felt and looked quite well. Her weight was then 73·3 kilograms (eleven stone nine pounds), the systolic blood pressure 135 and the diastolic pressure 92 millimetres of mercury, the resting pulse rate was 66 and the basal metabolic rate – 7·4%.

On January 7, 1926, the weight was 72 kilograms (eleven stone six pounds) and it remained at about this figure. On May 10, 1926, the basal metabolic rate was – 9%.

During the next two years the weight varied slightly, the maximum being 75·6 kilograms (twelve stone), but in April, 1928, she had an attack of acute tonsillitis, and despite an increase of thyreoid to 0·24 gramme (four grains) a day, the weight gradually rose to 80·1 kilograms (twelve stone nine pounds) in March, 1929. There were no symptoms of thyreoid insufficiency, however, and the same dosage was continued. The patient did not attend for two months after April, but then complained of slight palpitation which became so severe in August that she felt unwell. The resting pulse rate was found to be 96 per minute, and the weight had fallen to 70·6 kilograms (eleven stone three pounds). Thyreoid administration was stopped for one week and the basal metabolic rate was found to be − 2%. One week later she felt quite well and thyreoid medication was resumed, a start being made with 0·09 gramme (one and a half grains) a day. She gained 2·2 kilograms (five pounds) in weight, and then resumed her maintenance dose of 0·18 gramme (three grains) daily.

During the next winter 0·24 gramme (four grains) was given every day, but the optimum dose is now 0·18 gramme (three grains) a day. She consistently reports that she feels splendid, and on May 17, 1931, the systolic blood pressure was 148 and the diastolic pressure 86 millimetres of mercury, the pulse rate was 78 and the weight 82 kilograms (eleven stone six pounds).

The points of interest in this case were the rapid response to treatment in classical myxœdema, the small amount of thyreoid extract necessary to produce overdosage, the deleterious effect of an acute infection on the remnant of thyreoid tissue presumably present, slightly increased doses during the winter months, and the excellent results of treatment.

Mrs. H. McD., aged fifty years, attended at the hospital on June 21, 1926, with the history that, excepting for varicose veins, she had been well until December, 1925, when she had an attack of vomiting and diarrhœa. Shortly after this she noticed numbness of the left thumb, with pain which extended to the wrist. Swelling of the face and eyes became noticeable; she was always cold, and could not get warm. Her hair was falling out, memory was very poor and there was difficulty in walking. It was possible for her to do her housework, but her movements were very slow. Sleep was poor; appetite was capricious; bowel actions were normal. On waking in the morning she was unable to speak other than in a whisper. Menstruation had ceased five years previously. She thought that she had lost weight.

The typical facial appearance, slow speech, husky voice and dry skin of myxœdema were present, but there was no pseudo-œdema. There was some diminution of sensation over the dorsal aspect of the thumb and index finger of the left hand, apparently due to neuritis of the superficial radial nerve, but no evidence of other organic lesions was detected. The systolic blood pressure was 130 and the diastolic pressure 100 millimetres of mercury; the pulse rate was normal.

It was not possible to estimate the basal metabolic rate at the time, and thyreoid was given in 0·03 gramme (half grain) doses three times a day. In three weeks she lost 1·3 kilograms (three pounds) in weight, but felt no better, and the dose was increased to 0·18 kilogram (three grains) a day. Two weeks later she had greatly improved; speech was normal, she was walking much better and sensation was returning to the thumb. Her weight was then 66·1 kilograms (ten stone seven pounds). One month later her appearance had also greatly improved, but her hair was still falling and sleep and appetite were unaltered. Her weight was then 63·9 kilograms (ten stone two pounds), the systolic blood pressure was 135 and the diastolic pressure 95 millimetres of mercury; the pulse rate was normal.

For two years the patient continued to take the same dose of thyreoid, but then, for some unknown reason, it was reduced by half, though without any apparent ill effect. When, however, on November 7, 1929, her weight being 62·5 kilograms (nine stone thirteen pounds), the metabolic rate was determined, it was found to be − 48%. The dose was immediately increased to 0·18 gramme (three grains) a day for one month, and then to 0·36 gramme (six grains) *per diem*. After four months slight palpitation developed and the amount was reduced to 0·27 gramme (four and a half grains) a day. At that time the weight had fallen to 56·2 kilograms (eight stone thirteen pounds).

This dose was maintained until January, 1931, when the patient felt so well that she decided to discontinue both medicine and attendance at hospital. After four weeks she was unable to eat because of weakness of the jaws, she had no voice in the morning, memory was failing, hair commenced to fall, and she was unsteady in her gait. On resuming thyreoid medication she rapidly returned to normal, and has continued on her maintenance dose of 0·09 gramme (one and a half grains) three times a day. The basal metabolic rate with that dosage is + 6%.

The above case again illustrates the apparent ill effect of infective processes in damaging an inefficient thyreoid gland, the onset of symptoms being coincident with the occurrence of peripheral neuritis and probably attributable to an attack of gastro-enteritis. Other features of interest were the marked effects on the voice and gait and muscular power; also the poor response with inadequate dosage in the early stages, as opposed to the maintenance of apparent good health on the same inadequate dose which was given for eighteen months. It is remarkable that there should have been no signs or symptoms of myxœdema when the basal metabolic rate was − 48%. The relapse on cessation of treatment was characteristic, and it is worthy of note that the optimum dose was 0·03 gramme (half a grain) greater than in the first case. Further, it was shown that an increase of 0·09 gramme (one and a half grains) in the daily dose gradually resulted in toxic symptoms.

The above cases also present a marked contrast to those instances of subthyreoidism in which it is necessary to give enormous doses of thyreoid substance in order to produce any clinical effect. This difference is partly attributable to defective absorption in the latter cases, but in many of these the thyreoid deficiency is merely part of a polyglandular insufficiency.

Toxic Goitre.

Mrs. E.S., aged thirty-eight years, attended on February 4, 1931, complaining of an aching pain in the neck and right ear. She had been operated upon for pelvic trouble six months previously, and partial oophorectomy was performed. Following the operation the goitre, which she had known to be present for some years, became more marked, and the patient attributed the pain to this. She had not menstruated since the operation, but had continued to have a profuse vaginal discharge, due to endometritis and endocervicitis.

The thyreoid gland was irregularly enlarged, the right lobe being lumpy and apparently containing a large, firm, elastic, tender adenoma. No bruit was audible over the thyreoid, which moved freely. Tremor was marked, the skin moist, but there was no exophthalmos. The systolic blood pressure was 150 and the diastolic pressure 110 millimetres of mercury, the pulse rate was 84 per minute.

Unfortunately, she was given Lugol's solution (0·6 mil or ten minims three times a day) before the metabolic rate was estimated, and on March 7, 1931, the basal metabolic rate was + 14%. On April 21, 1931, when iodine had not been given for one month, the basal metabolic rate was + 44%, and the next day an attack of nausea and retching commenced and she felt and looked very ill. The pulse rate had risen to 105 per minute. Lugol's solution was resumed in doses of 0·6 mil (ten minims) thrice daily, and in one week she had greatly improved; the nausea had decreased, no vomiting or retching was present, and the pulse rate was 90, with a systolic blood pressure of 160 and a diastolic pressure of 92 millimetres of mercury.

After preliminary rest in bed, on June 1, 1931, she was operated upon by Mr. M. A. Stewart, who removed the adenomatous right lobe of the thyreoid.

Of particular interest in this case were: (i) the onset of toxic manifestations after an operation on the ovaries; (ii) gastro-intestinal crisis in a proved adenomatous goitre, with response to iodine medication.

Mrs. K.W., aged thirty-five years, was admitted to hospital on August 16, 1929, suffering from acute suppurative arthritis of the right ankle joint, due to infection with hæmolytic streptococci, and occurring two weeks after acute tonsillitis. The ankle joint was drained by Mr. Stewart, and a good recovery was made, except for a painful ulcer on the lateral malleolus. At that time there was a history of thyreoid enlargement for three years, but there were no toxic symptoms. There was a firm, rounded swelling in the left lobe of the thyreoid.

On April 10, 1929, the patient was readmitted with a history of nervousness, protrusion of the eyes and palpitation which had gradually become more pronounced since she left hospital.

There were present slight, but definite, bilateral exophthalmos, a widened palpebral fissure, tremor, sweating and tachycardia. On April 15, 1929, the basal metabolic rate was + 58%. She was given Lugol's solution (0·3 mil or five minims, thrice daily), with a resultant steady fall in pulse rate, and left hemithyreoidectomy was performed by Mr. Stewart on May 3, 1929. On May 21, 1929, the basal metabolic rate was +19%.

She had remained well and the basal metabolic rate on May 18, 1931, was + 14%. At that time there was a slightly widened palpebral fissure, but the patient looked well, and there was no clinical evidence of thyreotoxicosis.

This case was an example of the activation of an adenoma by sepsis, possibly aggravated by the stress of operation. The presence of exophthalmos in adenomatous goitre and its subsidence after operation are worthy of note.

The last case presented was a classical example of exophthalmic goitre.

Mrs. M.C., aged thirty-seven years, attended on March 6, 1929, complaining of tiredness, hot flushes, nervousness and loss of weight, which had been present for the preceding few months. Bilateral exophthalmos, tremor and tachycardia were present, with slight general enlargement of the thyreoid gland, over which a double bruit was audible. The tonsils were very large and septic. The classical picture of exophthalmic goitre was present, and after she had been made to rest in bed and had been given Lugol's solution, the right lobe, isthmus and pyramidal lobe were removed by Mr. H. R. Dew on April 9, 1929.

After an uneventful convalescence from this operation tonsillectomy was performed on May 2, 1929. Thereafter the patient was not seen until she reported on February 2, 1931, complaining of nervousness, tremor, tachycardia, dyspnœa on exertion, voracious appetite, loss of weight and sweating, which had been present for four months. During the preceding month there had been some pain over the precordium. Interrogation disclosed that her husband was out of work and that, in addition to great worry, she had been compelled to do hard physical work.

She was given Lugol's solution (0·3 mil or five minims, three times a day) and bromides at night to improve her rest, but would not consent to operation. On March 30, 1931, the basal metabolic rate was + 45%, the systolic blood pressure was 128 and the diastolic pressure 86 millimetres of mercury; the pulse rate while the patient was at rest was 120. Increased doses of iodine were given, but the patient could not rest and no improvement resulted. The systolic blood pressure on April 27, 1931, was 160 and the diastolic pressure 90 millimetres of mercury; the pulse rate with the patient at rest was 120. Again operation was urged, but refused. However, in May an attack of diaphragmatic pleurisy necessitated admission to hospital, and, despite this complication, rest and continued iodine treatment had resulted in marked improvement. There was no evidence of tuberculosis, either clinically or radiologically, and the patient was then awaiting operation.

The possible exciting cause of toxic symptoms in the above case was the presence of septic tonsils; but there was little doubt that the relapse was precipitated by worry and overwork. It illustrated the necessity of obtaining the patient's consent for a second stage operation whenever it is not possible to perform subtotal thyreoidectomy in exophthalmic goitre.

Discussion.

In the last of the above cases an attempt was made to gauge the metabolic rate by the use of a formula based on the pulse rate and pulse pressure, as it was not possible to have repeated instrumental estimations performed. For this purpose Read's second formula was used [B.M.R. = 0·75 (PR + 0·74 PP) − 72]. Though the estimation was made under conditions as near to standard as was possible in the out-patient department, it was realized that the figure was only an approximation to the actual metabolic rate. It was, however, felt that in the same patient the results obtained were an additional guide in determining progress or retrogression.

Recently Gale and Gale[1] analysed Read's series of cases and suggested another formula which gives promise of being a useful guide for the practitioner who is unable to have the basal metabolic rate determined by the usual methods. The error appeared to be much smaller than occurs with either Read's original formula or that mentioned above. They suggested the formula:

$$\text{B.M.R.} = (\text{PR} + \text{PP}) - 111.$$

As Gale's formula gave high results when the basal metabolic rate was below zero, Sharp[2] suggested that, if PR + PP was less than 111, the figure to be subtracted should be 111 + 5; if PR + PP was less than 91, the figure should be 111 + 10. This amendment gave more accurate results when applied to the appropriate cases in Read's series.

These cases supplied further evidence in support of Dunhill's[3] contention that the term toxic goitre is preferable to the word hyperthyreoidism. Cases of toxic goitre cannot be divided into two distinct groups, as suggested by Plummer, for it is possible to find all grades between toxic adenomata on the one hand and exophthalmic goitre on the other. The separation is warranted only in so far as it depicts the widely diverse types of toxicity that may occur, for, as Dunhill points out, the state of the gland at the time that it is subjected to a stimulus, and the nature of the stimulus, whether exciting or destructive, appear to determine whether toxic manifestations shall occur or not. If toxicity does result, the condition of the cells of the body, especially of the heart and nervous system, is also of great importance in determining the clinical type of goitre.

References.

[1] A. M. Gale and C. H. Gale: "Estimation of the Basal Metabolic Rate from Formulæ Based on the Pulse Rate and Pulse Pressure," *The Lancet*, June 13, 1931.
[2] B. Buckley Sharp: "Estimation of the Basal Metabolic Rate," *The Lancet*, June 20, 1931.
[3] T. P. Dunhill: "Toxic Goitre," *The British Journal of Surgery*, January, 1930.

Reviews.

EXERCISE FOR THE MIDDLE-AGED.

THE fifth decade as a rule finds the individual taking stock of himself and those who have arrived at the mid-term of life will find "How to Keep Fit After Forty," by Dr. Ronald Thornhill, a profitable investment.[1] It is a sensible little book, written in popular language, and deals with age and health, sunlight and fresh air, work, rest and recreation, diet, the digestive, respiratory and cardio-vascular systems, has a chapter directed to women, and concludes with a few illustrated exercises suited to the "decading." The advice given is non-controversial, wonderfully comprehensive and simply stated. It is a suitable book to recommend to patients with, in many cases, the assurance that attention to its counsel will not only give freedom from those subjective sensations so disturbing to the equanimity, but also restore their *élan*.

[1] "How to Keep Fit After Forty," by R. Thornhill, M.B., Ch.B.; 1931. London: Methuen and Company. Crown 8vo., pp. 126, with ten illustrations. Price: 2s. 6d. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 31, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE WASSERMANN TEST.

The quotation with which Dr. Cyril Shearman starts his special article in this week's issue, coming as it does from one of the latter-day giants in medicine, is worthy of much more than passing attention. It may be paraphrased by the statement that "to know syphilis is to know medicine." It is given to very few to know medicine. Twenty-five or thirty years ago the clinical manifestations of syphilis, seen in the out-patient departments of teaching hospitals, were very different from those seen today. The full bloom of the secondary rash is not often extant, mucous patches on tongue and lips and condylomata in the vicinity of vulva and anus are not so common, while rupial syphilis is almost a thing of the past. In spite of the characteristic signs and symptoms so often seen with syphilis of the olden days, diagnosis was not always a simple matter. The more rapid disappearance of the outward manifestations of the disease consequent on the use of arsenical derivatives in treatment, has not made its recognition at a later stage any easier. The Wassermann test is the main prop on which the clinician relies when he has reason to suspect the presence of syphilitic infection. To the laity the name Wassermann is fairly well known, and many people know what it means to "have a blood test." The non-medical person as a rule regards the test as infallible—he, of course, cannot be expected to know that it is not—and the medical practitioner has to be continually on the watch lest he attach too much importance to the catch phrase "Wassermann negative," so dear to the heart of the clinical pathologist. For this reason Dr. Shearman's article is commended to the careful attention of practitioners of all branches of medicine.

It is not necessary to traverse the ground that has been so well covered by Dr. Shearman. Two aspects may be emphasized. The first is the need for caution in the interpretation of persistent failure of the serum to react after treatment has been stopped. These are the circumstances in which most difficulty will be experienced. As Dr. Shearman shows, if the result of the test is regarded as a reflex of the production of reagin by the tissues in response to spirochætal activity, unwarranted assumptions are not likely to be made and the patient will not be left to the mercy of spirochætes which may be temporarily dormant or almost extinct. It also needs to be clearly understood—and Dr. Shearman has done well to emphasize it—that while a positive reaction to a Wassermann test is indicative of the presence of syphilis, it does not show that a particular lesion is syphilitc. In these circumstances great difficulty may arise, and even careful biopsy may not invariably dispel the doubt.

Since the Wassermann test is such a useful diagnostic weapon and therapeutic guide, the question arises whether it is fully exploited, whether sufficient search is made for the presence of syphilis. Syphilis appears in so many rôles and the stage is so carefully set that the syphilitic cloven hoof oft-times remains hidden. The cloven hoof must be sought. Perhaps in midwifery more than in any other branch of medicine is the routine use of the Wassermann test desirable. There would be very little difficulty about the introduction of the practice into obstetric hospitals; in private practice the difficulty would be greater, but not insurmountable. If all maternal and congenital syphilis were discovered and treated, the toll of disease in general

would be much less. In everyday practice syphilis must be sought as a basal or complicating factor in many conditions. Medical practitioners know full well that patients must be regarded from every aspect; they know that most people have at some period of their lives felt the desire for sexual indulgence and that inhibiting factors are not always strong; they know that the patient in later years forgets the indiscretions of his youth. No one must therefore be looked upon as immune. If medical practitioners make fuller use of the Wassermann test, they will learn more about syphilis and may eventually expect, as Osler foretold, the addition of all things clinical.

Current Comment.

CIRCULATORY MECHANISM OF MARATHON RUNNERS.

SINCE the days when the ancient Greeks instituted the Olympic Games in Athens, there has ever been a fascination, both to the lay and to the scientific mind, in a contemplation of mightly athletic effort. The layman, consciously or subconsciously, compares the feat with his own poor capabilities; the scientist ponders on the physiological effect of such great expenditure of muscular energy, the psychological factors involved and the many absorbing problems presented by the cardio-vascular system. It is held by some that voluntary athletic endeavour, however severe, does not cause permanent impairment of cardiac function, providing the heart be normal at the commencement. There are others who disagree with this view and who declare that such games as rowing and long distance running, which necessitate prolonged exertion and a great output of physical work, may endanger the future cardiac welfare of the individual, especially of the adolescent. The story is well known of the Greek courier who ran from Marathon to Athens and, crying "Rejoice! we conquer!" fell dead. Did this man possess a normal heart which, eventually from sheer exhaustion, ceased to beat? Or was it a diseased organ? It is difficult to imagine any but a highly efficient heart enabling a man to run a distance of 42 kilometres. But not every man possessing a normal heart, however highly trained he may be, can run and win races over long distances. The questions then arise, what manner of man is he who excels at marathon running? And in what manner do his heart and circulatory system differ from those of other men, that they allow him to perform with impunity such prodigious feats? The questions are discussed by Crichton Bramwell and Reginald Ellis, who made their observations during the progress of the Olympic Games at Amsterdam in 1928.[1] They examined over 200 athletes, of whom the runners were of the greatest cardiological interest. Runners may be divided into four classes: the sprinters, who race over distances of 100 and 200 metres; the middle distance runners (400 and 800 metres); long distance runners (1,500 to 10,000 metres); marathon runners (42 kilometres).

The men who run in the marathon race are older than other runners; they are of considerably lighter build, their pulse rate is slower and their systolic blood pressure a little higher. Bramwell and Ellis remark that men aged in the vicinity of thirty years possess greater stamina than men under twenty-five years of age. They are unable to determine whether this increased stamina is due to a nervous, circulatory or metabolic factor. They suggest that, as the older man's joints are less supple, he needs expend less energy in limiting their movements. As a consequence he runs more economically. The sprinter, on the other hand, must be supple. There is an important difference between the cardiac requirements of the sprinter and the marathon runner, for the former can run his race over one hundred metres without taking a breath, and, "so far as the immediate requirements of his muscles are concerned, he might almost dispense with his heart." But in the long distance races the circulatory mechanism must be highly efficient.

The question of oxygen available and oxygen required is an important one. If oxygen is used more rapidly than it can be acquired, a condition of oxygen debt is likely to occur. This results in breathlessness and greatly increased pulse rate. Marathon runners are not usually called upon to use an excessive amount of oxygen towards the end of their race as are runners in races over shorter distances. As a consequence they are not so likely to suffer from oxygen debt and to become greatly distressed.

Bramwell and Ellis draw attention to the possibility of the occurrence of hypoglycæmia at the conclusion of a marathon race. Two of the runners examined by them actually were affected with hypoglycæmia; they were cyanosed and very weak, and one had muscular incoordination. No cyanosis was observed in any man whose blood sugar content was normal. It is suggested that cyanosis is due to failure of the heart muscle on account of shortage of glycogen. Perhaps the runner of ancient Greece died in hypoglycæmic convulsions.

Bramwell and Ellis mention the heart ratio, that is, the relation of the weight of the heart to the weight of the body; they quote Clark's tables, in which animals with a high heart ratio are shown to be those capable of continuous and severe muscular exertion. When the width of the heart shadow as observed by X rays is plotted graphically against the body weight of athletes, the coordinates, excepting that representing the ratio for marathon runners, lie in fairly close proximity to a mean

[1] *The Quarterly Journal of Medicine*, April, 1931.

curve. The marathon runners appear to possess a definitely high heart ratio. Three individuals possessed hearts measuring more than fifteen centimetres in transverse diameter, though their average body weight was only 65 kilograms. One of these men finished second, another twelfth, and the third seventeenth in the race. Bramwell and Ellis admit that there are obvious fallacies in the measurement of the heart by radiological means, but they point out that their figures are averages of many measurements and that the same radiologist made all the examinations, using precisely the same technique throughout. They remark that a marathon runner needs a big heart, and it is probable that his heart enlarges as a result of prolonged training. But they suggest that perhaps he is born with a big heart and that this is his natural advantage and one of the attributes which make him able to excel at racing over great distances.

Electrocardiographic examination revealed that there was a right-sided ventricular predominance in 53 of 72 athletes of various classes. A notable feature was a prolongation of the *P-R* interval, which exceeded 0·2 second in seventeen instances. This, of course, is indicative of a delay in conduction from auricle to ventricle. In the electrocardiograms of seven men, five of whom were long distance or marathon runners, there was a remarkably high "take-off" of the *T* wave, resembling the appearance frequently seen in electrocardiograms of persons suffering from coronary occlusion.

An accentuation of the second cardiac sound, either at the apex or the base of the heart, was frequently heard, and in two of the marathon runners there was a reduplication of the first sound at the apex; in several others it appeared to be prolonged. In twelve athletes this prolongation was not unlike the crescendo presystolic murmur of mitral stenosis.

The marathon runners generally were of a more phlegmatic temperament than other athletes, and their pulse rates were less affected by emotion or other exciting factor.

There is a more or less prevalent opinion that men who indulge excessively in athletics, especially such severe athletics as marathon running, are in danger of storing up trouble for their later years, and that cardiac weakness from overstrain will eventually be their lot. It must be admitted that there may be danger in excessive exertion of any kind when the heart is diseased or is weakened by toxæmia, but it is difficult to find a reason why a heart which is now a highly efficient organ should be in special danger of becoming dangerously inefficient. Yet the term "athlete's heart" is not uncommonly employed and, to the ear of the layman, it has a terrifying ring. It is unlikely that the huge hearts of these marathon runners are pathological, for they remain unembarrassed throughout a race occupying two and a half hours, over a distance of forty-two kilometres.

It is to be hoped that these interesting studies of Bramwell and Ellis will be continued along similar scientific lines. What is required is a regular investigation of the cardiac function, together with a careful record of the illnesses and habits of the individual athlete and ex-athlete, entailing observation over a period of many years. By such means, perhaps, the ultimate effect of competitive athletics on the circulatory system and the decline, if any, in the efficiency of the hypertrophied heart could be gauged. The matter is of great importance in any English speaking community and of special importance to Australians.

JOHN MONASH.

The flight of time brings many changes. One by one the men who were leaders in the Great War are passing on to join their comrades who have "gone west." Now we mourn the passing of our greatest citizen soldier, Sir John Monash.[1] Full tribute has been paid to his work as a soldier, an organizer and a leader amongst men. There is one aspect of his work which, though not spectacular, played a great part in the success of his leadership. A leader is helpless without troops. It is not, perhaps, generally appreciated that the army is very dependent on the health of the troops. Monash was deeply appreciative of this basic principle. He was most attentive to all those things which contribute to the physical fitness and well-being of his men. He emphasized to his staff at all times his desire that his troops should be well clothed, well fed, and kept fit and happy. Those officers who were privileged to be in close personal contact with him, can bear this out. Monash realized better than most commanders the important place the medical services have to take with the armies in the field. He gave full support to every act and order which helped to keep the soldiers well or to care for the sick and wounded. His highly organized and well trained mind included in the scope of its activities the details of the medical care of the officers and men. He laid it down as a principle that the officer commanding the medical services of his division or corps should be kept fully informed of the intentions of the other branches of the staff. As a result the medical services under his command were happy and efficient. The officers of the Australian Army Medical Corps who knew Monash, found him a kindly gentleman, always courteous and considerate for others, demanding and expecting efficiency and full knowledge, but with a very human capacity for understanding. No one went to John Monash with a problem, military, medical or personal, and came away empty-handed. There are many medical practitioners who will remember the kindliness and thoughtful consideration of this leader, who was great not only in his powers of organization, his marvellous gifts of exposition and his qualities of leadership, but also in those truly human qualities of a courteous gentleman.

[1] In another place in this issue will be found a picture of the memorial bronze tablet sent by the officers of the Australian Army Medical Corps of New South Wales to the funeral of the late Sir John Monash.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Uterine Hæmorrhage.

BECKWITH WHITEHOUSE (*The Lancet*, January 3, 1931), in reviewing the general position of uterine hæmorrhage, with special reference to malignant disease, points out that during 1929 4,428 women died in England and Wales from malignant disease of the uterus. The average toll over the last five years shows that the death rate from this cause amounts to 215 to 220 per million women. In 30% of all women who suffer from cancer, the uterus is the first organ involved. He is strongly in favour of developing a cancerophobia, as it would save many lives if women who had excessive bleeding reported at once for investigation. He therefore reviews the whole question of hæmorrhage, particularly at the time of the menopause. Many hold that menstruation is the normal manifestation of two phenomena or factors A and B, which normally coincide. A ripe ovum is discharged from the ovary about the middle of each menstrual cycle during the reproductive age. From the *corpus luteum* which develops after the discharge of the ovum, a secretion is poured out which stimulates growth of the embryonic type of connective tissue cells of the endometrium, converting it virtually into a decidua ready for the ovum should it be fertilized. If the lutein hormone is withdrawn, the endometrium necroses with surface hæmorrhage. The lutein hormone is regarded as being antagonistic to ovulation, and so long as a normal and functioning *corpus luteum* is present, further development of Graafian follicles is, as a rule, suppressed. Should fertilization not occur, the stimulus to development of the endometrium is withdrawn by degeneration of the *corpus luteum* itself. If the *corpus luteum* is removed or destroyed, or even if the mature Graafian follicle is excised, cauterized or has its secretion aspirated by means of a syringe, the same thing may follow. The author states that it appears as though the mature follicular cell of the Graafian follicle produces at least one hormone identical with that of the fully developed lutein cell, but that in the fully developed lutein gland the secretion is more concentrated. Factor B is the influence of the hormone "œstrin" or the female sex hormone. The function of œstrin is apparently to produce a state of œstrus with general and local hyperæmia, increased secretion of the uterine glands, both of the body and cervix, and increased thyreoid activity. Factors A and B normally synchronize. The source of the sex hormone is obscure. It has been found in the placenta, in the urine of pregnant women and in many other tissues of the body. The sex hormone does not control the growth and activities of the ovum. There is absolute independence of ovulation and œstrus. In the human female both functions, whilst independent, are coordinated to promote the highest degree of fertility, but either in itself is sufficient to set up bleeding from the uterus. There is a definite curve of activity in relation to the sex function. The basal metabolic rate has been shown to divide itself into four phases corresponding closely to the various stages of endometrial development. Even when the uterus and ovaries are removed, the subsequent metabolic curve continues to show the usual menstrual disturbance, although the variations are not so obvious. At the actual menopause ovulation is commonly frequent, irregular and patchy, and this is reflected by variations in the metabolic rate. The author holds that a certain number of cases of irregular hæmorrhage are due to the occasional belated ripening of an ovum. In these circumstances the type of hæmorrhage, however, is, both in character and duration, very like that of an ordinary menstrual period, but the bleeding from the uterus in which there is a developing cancer is smaller in amount, bright red and more prolonged. It is a slight, irregular or continuous blood-stained discharge rather than a single, short, solitary hæmorrhage. Hence it is still just as important that any bleeding from the uterus after the menopause should be most carefully investigated.

The Sedimentation Rate Test.

A. MATHIEU, F. E. TROTMAN, H. D. HASKINS, E. E. OSGOOD AND JOYCE ALBERT (*American Journal of Obstetrics and Gynecology*, January, 1931) describe their technique and interpretation of two thousand one hundred sedimentation tests on one thousand one hundred and forty-five patients. In criticism of other methods they are of the opinion that a diluent militates against accuracy, and the constant observation of the technician over a lengthy period is undesirable. A modification of Westergren's method by H. D. Haskins and F. E. Trotman and E. E. Osgood was chosen. Pipettes graduated from 0 to 200 millimetres were used. The necessary one cubic centimetre of blood is collected in tubes containing two milligrammes of powdered potassium oxalate per cubic centimetre of blood. Two readings were taken, one at fifteen minutes and one at forty-five minutes. The distance traversed by the top of the red cell column in the thirty minute interval between the fifteen and forty-five minute reading was chosen as the best indication of the true sedimentation rate. However, in certain cases more information was gained when both fifteen and forty-five minute readings were recorded. Studies on two hundred healthy nurses gave an average rate of ten millimetres in the thirty minute period described; 90% of them were less than seven. A rate of above ten, even if the patient is menstruating, is to be considered as abnormal. It was found that unless there are other complications, the sedimentation rate in pregnancy remains normal until the third month. From this time on it increases and reaches its highest point at the tenth month, going from 10·3 to 40·5. In the uncomplicated puerperium it reaches a slightly higher level, 43·7, to become normal about the fourth week. When the puerperium was complicated by pelvic abscess, retained clots, pyelitis *et cetera*, the average was 75 millimetres. In gynæcological conditions it was found that all inflammatory conditions gave an increased sedimentation rate. In tubal pregnancy the rate was raised. In one hundred and thirty-eight patients with salpingitis, on whom five hundred and twenty-nine tests were made, the average rate at the first test was 42·3. Notwithstanding the average increase, there are too many normal or low rates to give the test much weight in the differential diagnosis between salpingitis and ectopic pregnancy. It is, however, of great value in determining the progress of individual cases. The authors are convinced that the operation for salpingitis does not give the maximum good results and safety to the patient unless it be done when the sedimentation rate has reached normal, and they feel that this test is definitely more sensitive than the temperature record or the leucocyte count in determining when the infection has become "cold." By this means their mortality rate has been reduced and post-operative convalescence shortened.

Tubal Sterilization.

H. FUCHS AND E. C. LORK (*Monatsschrift für Geburtshülfe und Gynäkologie*, June, 1931) have investigated by means of hystero-salpingograms the patency of the tubes after various methods of tubal sterilization. The method of crushing and ligating a portion of the tube was shown to be very unsatisfactory. In many instances the salpingogram showed that the injected fluid had escaped into the abdominal cavity through the tubes. The only method likely to be successful was excision of the tube. Even this technique showed some failures due to utero-peritoneal fistula formation. They therefore consider that the only safe method is to excise a wedge from the uterine cornua so as to include the interstitial portion of the tube in the excision.

Effect of Myomata on the Fœtus.

W. BUSCHKE (*Monatsschrift für Geburtshülfe und Gynäkologie*, August, 1931) summarizes the theories advanced regarding the effect of uterine fibroids on the development of the *fœtus in utero*. He gives the histories of three cases in which, except for the infants being rather small, no signs of congenital defects were observed. There was apparently no interference with placentation nor of deficient blood supply. He expresses

the opinion that possibly the anterior pituitary hormone, which is known to be secreted in excess during pregnancy and also in the presence of tumours, may have some effect in controlling the size of the fœtus. It is not clear whether the fibroid itself may not secrete some toxic substance which will prevent fœtal maturity. Further biochemical and animal experiments are essential to clear up this point.

OBSTETRICS.

Treatment of Puerperal Sepsis.

H. Ziegeler (*Deutsche Medizinische Wochenschrift*, April 24, 1931) gives his results with a new drug, "Euthagen," in the treatment of puerperal sepsis. It is a colloidal preparation containing 23% of silver and 7% of sulphur. After contact with the blood plasma the silver is apparently separated slowly in a metallic colloidal form. Five cubic centimetres of a 5% solution are given intravenously every second day for six injections. No after-effects were observed. The author used the drug in the treatment of 44 patients with severe infections; 26 died and 18 recovered. Sixteen of the deaths occurred in the first few days before the drug could reasonably be expected to exert any beneficial effect, and should be deducted. This leaves 10 deaths as against 18 recoveries. While regretting that the percentage of recoveries was not larger, the author is definitely of opinion that the injections prevented the development of generalized infections and that further experience should produce better results. The details and charts of several cases are given in detail.

Prolapse of the Cord.

W. Schiller (*Monatsschrift für Geburtshülfe und Gynäkologie*, May, 1931) has tabulated in detail the incidence and results of treatment of prolapse of the cord occurring at the Second Klinik at Vienna and at other centres. As a result of this investigation he maintains that when this complication may be expected to occur with *primiparæ* with contracted pelvis or *multiparæ* with oblique presentations, the safest place for delivery is in a well equipped hospital and not at home. Reposition of the cord is to be considered only with vertex or breech presentations, provided no pelvic contraction is present. After this is done uterine contractions are stimulated with pituitary extract while the fœtal heart is carefully watched. In contracted pelvis with *primiparæ* Cæsarean section should be considered if the fœtus be of importance, especially with elderly patients.

Dental Lesions During Pregnancy.

N. Temesváry (*Monatsschrift für Geburtshülfe und Gynäkologie*, April, 1931) states that the commonest dental lesion during pregnancy is hypertrophic gingivitis; less frequent conditions are dental caries, odontalgia and epulis. Various ætiological factors include excessive calcium drainage by the fœtus, alterations in the normal alkalinity of the oral secretions, local circulatory disturbances and endocrine dysfunction. Hypertrophy and hyperplasia of the gums are first noted, followed by secondary bacterial invasion and inflammation. Prophylaxis is most important, careful dental and oral hygiene and frequent visits to the dentist throughout pregnancy being necessary. While extraction is to be prevented as far as possible, such treatment has no effect on the continuation of pregnancy, except during the fourth, eighth and twelfth weeks, when it should not be carried out. He advocates the establishment of dental clinics at all obstetric hospitals and also believes that the nurses should undergo regular dental attention. Care of the teeth will eliminate a fruitful source of endogenous infection during the puerperium.

Treatment of Asphyxia of the New-Born.

Louise McIlroy (*The Practitioner*, June, 1931) suggests a routine treatment of *asphyxia neonatorum*. In treatment of *asphyxia livida* the author suggests that the mucus should be sucked out, using a number 11 catheter with a pipette bulb in the middle. In mouth-to-mouth insufflation there is a danger of infection and excessive pressure on the alveoli. If respiration does not occur, a mask to which is delivered a mixture of 95% oxygen and 5% carbon dioxide at a pressure of eight millimetres of water and at a rate of two litres per minute is placed over the child's mouth, about sixteen times a minute. If breathing is unduly delayed, one cubic centimetre of lobelin is injected. The cord is cut and induced to bleed to relieve the strain on the right heart. No artificial methods of respiration involving movements of the head or spine are employed, nor any type of baths. Gentle massage of the heart or compression of the ribs may be carried out and the mucus extractor again used, if necessary. If respiration fails to take place, tongue traction is employed. It is stated that this is rarely necessary when carbon dioxide and oxygen are available. In the treatment of *asphyxia pallida* the same routine is carried out, except that a cardiac stimulant, such as camphor, is given. The baby is to be handled with the utmost care. Warmth and rest are absolutely necessary. Carbon dioxide and oxygen are used when the heart condition improves.

D. P. Murphy, R. Wilson and J. Bowman (*American Journal of Obstetrics and Gynecology*, April, 1931) report on the treatment of thirty-five asphyxiated new-born infants. The apparatus is a metal chamber in which the patient is placed with the head protruding through a snugly fitting collar. The chamber can be kept at any desired temperature and by means of a pump the air pressure is alternatively decreased and then allowed to return to atmospheric pressure at the rate of thirty-five per minute. The authors claim that by this apparatus they have been able to resuscitate infants with a very severe grade of asphyxia. They lay stress on the point that the air passages must first be cleared. Once the child commences breathing the machine is stopped, as the alternating pressure has no longer any effect on the respiratory movements. It was noted that a far higher percentage of success was obtained with those patients on whom treatment was commenced within a few minutes.

Abortions in Russia.

F. T. Taussig (*American Journal of Obstetrics and Gynecology*, July, 1931) gives an account of his impressions whilst on a visit to Russia. He inspected the abortaria in which 40,000 abortions are done in Moscow each year. There are two of these institutions in Moscow. The stay of the patient in hospital is about four days. A certain number of beds are free, but the majority of patients pay at the rate of one twenty-fifth of the average family income. Practically any woman can have the operation done, but the following classes are eliminated or reduced to a minimum: *Primiparæ* who can give no valid reason why they should not have a child; women who are past the third month and those who have infected cervices. The operation is done without anæsthesia. The procedure is to dilate the cervix and curette the contents out. A claim for zero mortality is made. There is, however, evidence in the Russian literature of the harmful psychic effects, gynæcological lesions and secondary sterility coming as an aftermath to these procedures.

The Conservative Treatment of Ablatio Placentæ.

G. Kornfeld (*American Journal of Obstetrics and Gynecology*, July, 1931) found fifty cases of *ablatio placentæ* in 1,600 confinements. There are many terms applied to the premature separation of the normally situated placenta, the most common being accidental hæmorrhage, utero-placental apoplexy, *abruptio placentæ* and *ablatio placentæ*. The condition is usually associated with a toxæmia, the most common being chronic nephritis. In the author's series, which was without a maternal death, Cæsarean section was done only once and only in the belief that a *placenta prævia* was being dealt with. Shock was treated with morphine and warmth. Contractions were induced with two to three minims of pituitrin and in some cases simple rupture of the membrane was sufficient; a bag was used in two cases. The author claims that conservative treatment gives far better results than the more radical measures advocated by some authors.

Special Articles on Aids to Diagnosis.

(Contributed by Request.)

VI.

THE CLINICAL SIGNIFICANCE AND INTERPRETATION OF THE WASSERMANN REACTION.

Osler sagely remarked: "Know syphilis in all its manifestations and all other things clinical will be added unto you." This disease is so protean in its symptomatology and is further characterized by the fact that it may remain asymptomatic for so long and so indefinite a period that clinical recognition of the condition is often extremely difficult, if not impossible. For this reason and because of its proven value, the serological examination of the blood for evidence of syphilis has become one of the most frequently used of laboratory tests.

In order to assess the full value of the test and its uses and limitations in clinical medicine, the clinician no less than the pathologist must know when and under what circumstances a reaction to the test may be expected and to be able when necessary to account for a failure to react even in undoubted syphilis. It must also be remembered that the significance of the result cannot be dissociated from either the reliability of the technique employed or the competence and experience of the serologist.

In discussing the significance and clinical interpretation of the result of the test, consideration must be given to the extent of treatment, if any, that has been administered, and to the stage of the disease existing or suspected when the test is made. We may then consider the findings under two headings: (i) untreated syphilis and (ii) treated syphilis, and we may discuss these in respect of the following types of the disease: (*a*) primary syphilis, (*b*) secondary syphilis, (*c*) tertiary or chronic syphilis and (*d*) congenital syphilis and syphilis complicating pregnancy.

Primary Untreated Syphilis.

Speaking generally, the property of giving rise to a positive reaction is not acquired by the serum until the late period of the primary stage. A positive reaction is regarded as being due to the presence in the serum of "reagin" which is produced as a result of tissue reaction against spirochætal activity and until reagin is present in detectable amounts the result of the test will be negative.

Boas concluded from his own and other observations in the literature that a positive reaction may be obtained at the earliest in the seventh week after inoculation and usually not before the ninth week. Kolmer finds that 44% of patients with primary syphilis give a positive reaction within five to fourteen days of the appearance of the sore, while 94% give a positive reaction four to six weeks after the sore has appeared. Kilduff states that the usual period at which a positive finding may be expected is from ten to twenty days after the first appearance of the chancre. My own experience leads me to expect a positive result in from two to three weeks after the appearance of the sore and usually in from five to eight weeks after the time of infection, the length of time varying inversely with the degree of tissue reaction at the site of inoculation. I have seen one case in which a positive response occurred on the twenty-eighth day after infection. A negative response in the early stages of possible infection must not then be regarded as evidence that syphilis can be excluded; the test should be repeated at regular intervals up to at least eight or ten weeks after a suspicious sore has first been noted. A partially positive reaction in the primary stage affords strongly presumptive evidence that the lesion is syphilitic, and if on repetition the reaction is found to be stronger, definite confirmative evidence of the nature of the lesion is afforded.

Secondary Untreated Syphilis.

In untreated secondary syphilis the Wassermann test gives a positive reaction in practically 100% of cases. Craig reports 96% of positive results in this stage, and Browning and McKenzie 95%. With the more sensitive methods of technique now in use it is rare to find a case of secondary syphilis which, before treatment has been instituted, does not yield a strongly positive reaction to the test.

Untreated Tertiary Syphilis.

In untreated active tertiary syphilis positive reactions, according to Kilduffe, are found in about 96% of cases. Occasionally in lesions affecting the *septum nasi*, as well as in some tertiary syphilides, the patient's serum fails to react. This is probably associated with a poor vascular supply to the part affected. If tertiary syphilis is suspected and the blood gives no response to the Wassermann test, the test should be repeated after the administration of a provocative dose of an arsenobenzene salt. In certain cases such a provocative injection will transform a negative into a positive response—the so-called Jarisch-Herxheimer reaction.

In neurosyphilis the blood serum may not react to the Wassermann test, but the cerebro-spinal fluid yields a positive reaction in practically every case and, failing other evidence, lumbar puncture should never be neglected if neurosyphilis is suspected.

It is characteristic of syphilis that the symptoms tend to disappear spontaneously at certain periods. Ehrlich describes this as being due to a non-sterilizing immunity which may occur apart from any specific treatment. In these so-called latent periods of untreated tertiary syphilis the blood fails to react to the Wassermann test in almost 50% of cases, but some of these can be "provoked" to give the Jarisch-Herxheimer reaction.

Kilduffe states that latent and asymptomatic syphilis, the only evidence of which is a positive Wassermann reaction, is more frequent than has hitherto been appreciated, and without question has been responsible for many of the so-called "false positive reactions" reported in the earlier serological studies of health and disease. Unsuspected positive reactions, that is, reactions occurring in patients who show no clinical evidence of syphilis, are met with in about 3% of the adult population.

Before acquainting the patient with the result, it is always advisable in such cases to confirm it by repeating the test with fresh blood, since "false" positives may occur as a result of faulty technique and since serologists, like clinicians, are not infallible.

If on repetition, the reaction is again strongly positive, it is safe to assume that the patient is suffering from latent syphilis. The only other diseases that are definitely known to cause the blood to react to the Wassermann test, that is, to yield "false" positive reactions, are yaws (*frambœsia tropica*) and gangosa (tropical ulceration of Guam and the Philippines).

Kolmer has shown that with the recent improvements in technique such diseases as malaria, leprosy, diabetes and the various febrile disturbances that were previously stated to yield "false positives" do not cause the patient's blood to react, except in the presence of syphilis.

In the hands of a competent serologist using any of the approved modern methods of technique false positive reactions are rarely encountered. If a reaction is positive when syphilis is unsuspected, the test should always be repeated to exclude the possibility of technical error. A false negative response may occur in some cases of tertiary syphilis and in latent syphilis, but if the disease is suspected and no reaction occurs, the test should always be repeated after a provocative injection of an arsenical salt.

Untreated Congenital Syphilis and Pregnancy.

In children born with obvious stigmata of syphilis, the reaction is positive in practically 100% of cases. In children born of syphilitic mothers, but showing no stigmata, positive reactions are observed in from 70% to 80%, if the test is carried out six to eight weeks after birth. Immediately after birth or within the first week or two, no reaction may occur, and for this reason, if syphilis is suspected, the child's blood should again be examined

after the lapse of a month or six weeks. Reactions with the blood from the cord are apt to be unsatisfactory, being frequently anti-complementary and, according to some observers, non-specific reactions may at times be observed.

A negative response with the cord blood is inadequate evidence that syphilis may be excluded from either mother or child. A positive reaction with the cord blood is regarded by some authorities as evidence of syphilis only in the mother, but, according to Kolmer, should be regarded as indicating infection in both mother and child. The same authority recommends making at least two routine examinations of all children born of syphilitic parents, namely, (*a*) the first within a few days to one week of birth and (*b*) the second four to eight weeks later. The result of the first examination may be negative and of the second positive or *vice versa*. In either case he regards the child as potentially syphilitic and this is a certainty if both results are positive.

Pregnancy exercises a modifying effect on the course of syphilis, and as a result the manifestations of the disease may be suppressed or so altered and delayed that, even with the most careful examination, they escape detection. This fact was responsible for the enunciation of Colles's law and Profeta's law, both of which have been shown by the application of serological examinations to the study of such cases to be based on a false assumption. Owing to the modifying effect exerted by pregnancy on the course of the disease, variations in the serum reactions also occur during this period.

A positive reaction in the maternal serum during pregnancy is of undoubted significance, but a certain percentage of cases, even though showing the stigmata of secondary syphilis, fail to yield a response to the test. Absence of reaction therefore in pregnancy is of doubtful value in excluding the possibility of syphilis. If such possibility exists, the test should be repeated both before and after delivery.

In untreated syphilis, whatever phase of the disease is present or suspected, the Wassermann test is used as a confirmatory aid to diagnosis, and in evaluating the response the necessity of combining clinical with serological evidence must be stressed.

With a modern and approved method of technique, and in the hands of an experienced and careful serologist, a positive reaction, even though the disease be unsuspected and asymptomatic, must be regarded as affording strongly presumptive evidence of the presence of syphilis. With corroborative clinical findings, a positive reaction affords definite evidence of spirochætal activity. It must be remembered, however, that while positive findings confirm the presence of syphilis, they do not necessarily indicate that a particular lesion is syphilitic, so that failure of a lesion to respond to antispecific therapy does not necessarily imply that the blood reaction was false. The strength of the reaction is in proportion to the degree of spirochætic activity and the degree of tissue reaction to such activity. A partially positive reacton should not be regarded *per se* as either evidence of syphilis or, if it is not very strong, that the disease may be excluded. The result should always be confirmed by repetition of the test, and if necessary after a provocative injection has been given.

The detection of small quantities of "reagin" due to spirochætic activity varies with technique and with the activity of the reagents used in the test, so that it may be impossible ever to render any serum test sufficiently sensitive to detect all extremely quiescent or latent infections. The significance of this is that a single failure to react should never be accepted as definitely excluding syphilis when suspicious clinical signs or history are present.

Treated Syphilis.

There is now abundant experimental and clinical evidence to show that a positive reaction during the course of treatment is significant of the presence of living spirochætes and indicates the necessity for continuance of treatment. The lesions, however, are not necessarily obvious, and it must be remembered that occasionally in active syphilis, especially during treatment, the serum may yield no reaction to the test and also that a single negative response is no evidence that the condition has been cured.

In so-called "Wassermann fast" cases in which the serum yields a persistently positive reaction in spite of treatment, the bulk of evidence now points to the fact that a focus (or foci) of living spirochætes still remains. Fordyce states that necropsy in a series of such cases showed the lesion to be in the aorta in 90% of those examined.

As would be expected, the earlier in the course of the disease treatment is commenced, the more marked will be the influence of treatment as indicated serologically. Some cases of primary syphilis in which diagnosis is made by the dark ground microscope and in which treatment is instituted before the serum yields a positive reaction, may never yield a positive reaction throughout the course of the disease. If not positive before treatment is commenced, the response frquently becomes positive at a later date, even when treatment has been instituted, but with persistent treatment the reaction usually becomes weaker during the first year until it fails to occur.

It is usual to regard as one of the most valuable indications of cure a blood reaction remaining persistently absent for two years after all treatment has ceased. If we regard the response to the Wassermann test as evidence not only of infection, but essentially of tissue reaction to spirochætic activity whereby "reagin" is produced, it is obvious that caution is necessary in declaring any patient to be permanently cured. A negative response to the test affords evidence that at the time of testing there is insufficient interaction between tissues and spirochætes to produce reagin in serologically detectable amounts. Bearing in mind the potentiality of the disease to remain latent and asymptomatic for a period during which reagin may be produced in amounts that cannot be detected serologically, can we assume that any particular number of failures to react definitely proves the absence or cure of infection? Kilduffe regards repeated absence of reaction as evidence only of probability of cure, and the longer the period of time without treatment and without symptoms during which reactions cannot be obtained, the greater becomes the probability of cure.

In view of the possibility of early invasion of the nervous system, many authorities advocate that no patient should be dismissed from observation in spite of repeated failure of the blood to react, until the cerebro-spinal fluid has been fully investigated and found free from reaction also. Kilduffe sums up the value of the negative response during treatment as follows:

> It is to the ultimate benefit of the patient that he be taught at the onset of his infection that continued vigilance is the price of safety and continued observation a *sine qua non* of the treatment of syphilis. A negative reaction is merely an indication of *status presens*. It bears no guarantee for the future and holds no promise of permanency.

He suggests that a safe routine plan to adopt after a period of two years without treatment and without symptoms and with absence of reaction to blood tests, is to advise that the test be repeated once a year during life. His view is that failure to react after treatment should be regarded as merely an incident in the course of a search for a possible recurring positive reaction.

While this may be counsel of perfection, to many patients it would constitute a counsel of despair, especially in respect of a disease which responds so readily and favourably to treatment, so that many clinicians may regard the standard as being unnecessarily high. It must be remembered, however, that this high standard is not altogether at variance with pathological and *post mortem* findings.

Cyril H. Shearman,

Serologist, Sydney Hospital; Honorary Pathologist, Lewisham Hospital.

British Medical Association News.

SCIENTIFIC.

A meeting of the Section of Medicine of the New South Wales Branch of the British Medical Association was held at the British Medical Association House, Macquarie Street, Sydney, on September 17, 1931, Professor C. G. Lambie, the Chairman, in the chair.

Diabetes.

Dr. L. W. Dunlop read a paper entitled: "Some Aspects of Diabetic Coma and Arteriosclerosis" (see page 533).

Dr. C. H. Shearman demonstrated some lantern slides illustrating the histological appearances of arteries affected with degeneration apparently due to diabetes.

Dr. E. B. Jones demonstrated an apparatus designed for use in the estimation of the carbon dioxide combining power of the blood plasma.

Dr. R. Coupland Winn, in opening the discussion, remarked that there was no need for him to congratulate Dr. Dunlop on his excellent paper, or Dr. Shearman and Dr. Jones on their demonstrations; congratulations were superfluous. He would like to place on record, however, an expression of his gratitude to Dr. Dunlop for his advice in connexion with a case of diabetic coma that had been under Dr. Winn's care at Sydney Hospital recently. He also wished to take the opportunity of mentioning the devotion to duty shown by the resident medical officers of that institution in making hourly estimations of the blood sugar content until nearly four o'clock in the morning. It seemed to him that the public should know how hard the medical officers of a general hospital had to work at times. There were three practical points in the treatment of diabetes that he wished to discuss. Though Sansum's view that arteriosclerosis could be prevented by giving a diet containing a high proportion of carbohydrate had not been proven, and there was no final proof of the theory that foods with an alkaline ash were of prophylactic value, it seemed to him that these two possibilities should be taken into account. The first practical point that he wished to raise then, was that, if it was decided to give a high carbohydrate diet, the cost of the large quantities of insulin required might be prohibitive for all save a wealthy few; though perhaps many would be able to afford the smaller quantities of insulin required when a medium carbohydrate diet was being taken. For his own use he had devised a diet system that permitted him to prescribe diets in keeping with the amount of insulin each patient was able to afford. For instance, if a patient required a diet possessing a caloric value of 1,500, 64, 105 or 150 grammes of carbohydrate could be prescribed, according to whether he was able to afford a small, medium or large quantity of insulin. By means of the "diet group" system that he had devised, diets of these three types could be written up on a card, such as a small index card, so that by varying combinations of the groups, which consisted of 500, 600 and 900 calories respectively, diets might be prescribed consisting of any number of hundreds of calories from 1,000 to 4,000. These diets comprised foods rich in alkaline ash. The second practical point was that ordinary diets might be unsuitable for a patient suffering from diarrhœa. By means of the "group" system, soft diets of low, medium and high carbohydrate content containing any number of hundreds of calories, could be prescribed offhand. The third practical point depended on the discovery of Jonas and others that the blood sugar content could be kept nearest the normal by giving two doses only of insulin during the twenty-four hours. A double dose was given before breakfast and a normal dose before the evening meal; the patients were thus freed from the discomfort of the injection before the midday meal. Incidentally, Jonas and others gave, in severe cases, a dose of insulin at midnight, when the patients were in hospital, in order to bring the blood sugar content to normal before breakfast.

Dr. Sinclair Gillies congratulated Dr. Dunlop on his excellent paper. He thought that in regard to the causation of arteriosclerosis, specialism in diabetes had perhaps caused some modern observers to be led astray. He asked was there any definite tangible evidence that diabetes could cause arteriosclerosis? Granted that the existence of an increased blood sugar content was diagnostic of diabetes, the question might be asked, what was the cause of the diabetes? Certainly, in middle-aged people especially, diabetes and arteriosclerosis existed; but was it not possible that the arteriosclerosis caused the diabetes or that they arose from a common cause? He thought that this possibility might be overlooked by some recent observers. He doubted whether the causal relationship of diabetes to arteriosclerosis could yet be regarded as proven. Dr. Dunlop had mentioned vomiting as a symptom of diabetic coma; his own experience was that vomiting as an early symptom was uncommon. The possibility should not be overlooked that the onset of coma might be precipitated by the vomiting. Dr. Gillies mentioned the acidosis that so commonly occurred in children as a result of gastric disturbance. Perhaps a similar upset precipitated an acidosis and coma in diabetes. Dr. Dunlop's suggestion that the cardiac seizures suffered by one of his patients after giving insulin were due to a lowering of the blood sugar content was interesting; but it was doubtful if this was the correct explanation. It appeared to him that possibly there had been a psychical influence. He wished to ask what was the exact action of insulin? There seemed to be an attitude on the part of the medical practitioner to expect insulin to do just what he wanted it to do at the moment and to blame it where it failed to relieve coma when given in massive doses, irrespective of what other factors might be present.

Dr. G. C. Willcocks asked whether Dr. Dunlop considered that glucose should be given as a routine in the treatment of diabetic coma; if so, in what dosage per unit of insulin should it be given? Should it be administered by mouth, by rectum, intravenously or subcutaneously? He had given glucose along the lines recommended by the authorities, but his results had been unsatisfactory.

Dr. E. W. Fairfax expressed the opinion that it was not a good thing to give glucose in the treatment of diabetic coma until there was a decided fall in the blood sugar content. He believed that, if very large quantities of fluid were given and elimination was not good, the circulation might be overstrained, with the result that the lungs became œdematous. He thought that in some of the cases he had seen, the administration of excessive quantities of fluids might have been the cause of the failure of the circulation. He was unable to agree with those people who believed that the arteriosclerosis in diabetes was due to increased quantities of cholesterol in the blood; in nephrosis there was a high blood cholesterol content, yet it was not associated with arteriosclerosis. There must be some factor other than high blood cholesterol.

Dr. R. J. Millard expressed his thanks for the paper and demonstrations. He thought that when the advisability of giving glucose in the treatment of coma was being considered, the question should be asked, what was the cause of the coma? Was it of greater importance that there was a high content of blood sugar or of other substances in the blood? What was required was an increase in the carbohydrate metabolism. It did not matter so much that there was a high blood sugar content, but it did matter that there were present the harmful products of faulty fat metabolism that were causing symptoms. For his part he felt more comfortable if glucose were given. He agreed with Dr. Fairfax that harm might be done by the administration of too much fluid. Joslin did not believe in giving a great deal of fluid intravenously on account of the danger of too much strain being thrown on the circulation. An old woman on the verge of coma had been admitted to the Coast Hospital during the week. On admission her blood contained 420 milligrammes of sugar per 100 cubic centimetres. She was given 40 units of insulin and, during the night one pint of fluid by mouth. She passed no urine during the night. In the morning 738 cubic centimetres (twenty-six ounces) of urine were withdrawn by catheter; it contained about 1% of sugar; but the blood sugar content had fallen to 35 milligrammes

per 100 cubic centimetres and she was unconscious and cyanosed. Two pints of saline solution containing gum and glucose were administered intravenously, and the blood sugar content at the end of this injection was 141 milligrammes per 100 cubic centimetres. She had by then wakened and answered a few questions, but she died one hour afterwards. He had thought ever since that perhaps she had been, so to speak, drowned and that possibly she would have recovered if glucose only and half the quantity of saline solution had been given. He had seen another woman in coma recently. She had been given one and a half pints of saline solution intravenously, and she had recovered rapidly from the coma; but she was a bigger woman. The administration of epinephrine should be considered in these cases, for in acidosis and coma the pulse was of a very low tension.

DR. E. H. STOKES remarked that he had seen two of the patients mentioned in Dr. Dunlop's paper. The patient who had suffered from heart block while receiving treatment, had been sent to the Sydney Hospital from the Eye Department on account of arteriosclerotic changes observed in the fundus; these had been the first recognized signs of failing health. The thirteen year old boy mentioned by Dr. Dunlop had had albuminuria, and the cholesterol content of his blood had been 196 milligrammes per hundred cubic centimetres twelve months before the onset of coma. The blood cholesterol content of the patient whose X ray films had been shown, had been 424 milligrammes per hundred cubic centimetres. The figures were perhaps of interest, as cholesterol had been mentioned as a possible factor in the causation of arteriosclerosis.

DR. ROBERT S. STEEL said that he regarded diabetic coma at a slightly different angle; namely, from the viewpoint of the general practitioner. Dr. Dunlop had shown that in the past few years the numbers of fatal cases of coma had decreased greatly, and he had suggested that this had been due to better treatment received by the patients outside the hospitals. Dr. Steel thought that the general practitioner was still afraid of the use of insulin; if he could gather more confidence, the patients admitted to hospital would be in much better condition. The general practitioner was perhaps afraid of hypoglycæmia; but there was little need to be afraid of the development of this contingency. The point should be stressed that while the urine contained no sugar coma could not occur. In Dr. Steel's opinion coma was almost invariably preventible. The most important preventive measure was the education of the patient in dietetics and the use of insulin. In regard to the administration of glucose to patients suffering from coma, he pointed out that F. N. Allan, writing in *South-western Medicine* recently, had expressed the opinion that there was no need for hurry in giving glucose, that it was useful later in the treatment, but not in the beginning.

DR. O. A. DIETHELM congratulated Dr. Dunlop on his paper, which contained a wealth of material of practical importance. He thought that when large and massive doses of insulin were given in the treatment of diabetic coma, either at one time or repeated at short intervals, the fact should be stressed that insulin reactions might occur; they might occur also when the blood sugar content was too quickly reduced from a very high level to a level which was still well above the level at which insulin shock might ordinarily occur, say, from 0·4% or higher to 0·15% or so. Consequently it was safer to give moderate doses repeated at suitable intervals; the results obtained were just as satisfactory. Elderly people with advanced arteriosclerotic degeneration might suffer from all types of diabetes; but there was an inherently mild type of diabetes observed in arteriosclerotic persons of either sex (usually men fifty to sixty years of age) who had a history of glycosuria for varying periods of ten, fifteen or twenty years or longer. These patients did not have such a pronounced inability to burn up glucose as had other diabetics. Some arteriosclerotic diabetics receiving a good maintenance diet continued to pass in the urine a small amount of glucose, say 0·5% to 1% or a little more. To reduce the urinary sugar to nil by reducing proportionately the carbohydrate intake seemed quite an easy matter; but it would be found that when this intake was reduced, even to much lower limits, the patient would continue to pass the same amount of glucose in the urine; that was to say he might pass the same amount when taking a diet with a glucose value of, say, 160, as when taking a diet with a glucose value of even half this amount. In these cases the medical practitioner should pause and consider whether it was not better to prescribe a good maintenance diet and allow the patient to pass a little glucose in his urine and at the same time maintain his nutrition than to reduce his diet to such an extent, in trying to keep his urine sugar-free, that his strength and ability to get about would be affected. Dr. Diethelm said that occasionally it would not be possible to keep the urine of these patients sugar-free, completely and constantly, even with the aid of insulin, without doing harm and without a necessary reduction in the diet of such an extent that it would not be sufficient in caloric value for even a basal diet; moreover, to do so was unnecessary and illogical.

DR. C. H. SHEARMAN remarked, concerning Dr. Fairfax's objections to the theory that arteriosclerosis was due to cholesterol excess, that the xanthomata of diabetes consisted of deposits of cholesterol. Was it not possible then, that cholesterol might be deposited in the arterial walls in a similar manner? He had not seen as many cases of nephrosis as he had of diabetes, but he had never observed xanthomata in the former condition.

DR. ALLAN S. WALKER remarked that Joslin had laid down certain histological criteria that established the pathological diagnosis of arteriosclerosis in cases of diabetes. But it was very difficult to lay down any clinical criteria. Until it was possible to say from clinical examination that arteriosclerosis was or was not diabetic in origin, or even that it was present and to what degree, there was sure to be confusion. The diverse views that had been expressed concerning the treatment of coma made it clear that there could be no hard and fast rules and that each patient had to be treated individually according to his needs.

DR. W. E. FISHER said that among Dr. Evans's outpatients at Sydney Hospital, several middle-aged diabetics reacted to the Wassermann test. It was known that syphilis was a cause of endarteritis. Bearing in mind these cases, he was the more inclined to agree with Dr. Gillies's suggestion that in elderly arteriosclerotics the vascular condition should be considered as a cause as well as an effect.

DR. E. B. JONES expressed the opinion that, in a big hospital where facilities were available for estimating the blood sugar content, it was unnecessary to administer glucose until the blood sugar content had fallen to 300 milligrammes per 100 cubic centimetres.

PROFESSOR C. G. LAMBIE said that very few remarks had been made in the discussion concerning diagnosis, though this had been a feature of the paper. Intracranial disease had to be considered in the diagnosis of diabetic coma. On the one hand cerebral hæmorrhage might be a complication of diabetes; on the other, cerebral hæmorrhage from some other cause might give rise to glycosuria and even acetonuria. One patient whom he had seen, had become comatose as a result of cerebral hæmorrhage and later had developed diabetic coma. In another case of coma there had been little of diagnostic value to make out by clinical examination; but when consciousness had returned, an examination of the fields of vision had revealed the existence of a hæmorrhage in the right occipital cortex. In another instance there had been a cerebellar hæmorrhage. These patients had all improved considerably, but had ultimately died of cerebral complications. It was of interest to note that hæmorrhages resulting from diabetes did not occur in the usual regions; they had occurred commonly in the occipital lobes.

In the matter of treatment the questions had arisen whether glucose and a lot of fluids should be given. The two questions were bound up with one another. In many instances the patient was suffering most from anhydræmia and fluid had to be administered; but this was often overdone. There was a definite danger of œdema of the lungs or circulatory failure if large quantities of fluids were given

intravenously. On the other hand, very large amounts could be given subcutaneously with impunity. The administration of too much glucose made the blood more and more hypertonic and thus might aggravate the symptoms of anhydræmia. The estimation of the blood sugar content was of doubtful value as a guide to treatment; it might even be fallacious. If it was not known whether or not the patient had glycosuria, a knowledge of the blood sugar content might lead to the drawing of erroneous conclusions. He had seen patients with a high renal threshold who, when their blood sugar content had been reduced to 400 milligrammes per 100 cubic centimetres, had suffered from insulin shock. If insulin was given and the urine was not examined regularly for the presence of sugar, insulin shock might occur, though the results of blood sugar estimations led the physician to believe he was on the safe side. The examination of the urine was more important than the examination of the blood. The pulse and respiration rates were also better guides. If the patient had been otherwise improving and his pulse rate commenced to rise, the possible onset of insulin shock should be suspected.

The actual cause of the arteriosclerosis of diabetes was still unknown. All suggestions were in the region of speculation and all theories seemed to break down when submitted to a closer examination. There were many possible causes. The arteriosclerosis of diabetes might be a specific type of arterial degeneration. The idea could be dismissed that syphilis was an important cause of glycosuria; there was no real correlation. Arteriosclerosis could occur in young patients with diabetes of definite pancreatic type. It was not impossible, however, that the arterial disease of the so-called gouty glycosuria was the primary condition and the glycosuria secondary. Were there two different types of individuals to be considered, or were there two different ætiologies? The answer was not forthcoming. The younger patient and the thin diabetic were those who appeared to be most likely to die of coma; they required insulin to control their glycosuria. The fat type of diabetic did best by taking a reduced diet and often required no insulin, though, of course, it was always possible that the fat diabetic might become of the thin type. For this assertion there was no less an authority than Shakespeare: "At first the infant, muling and puking in its nurse's arms"—this child obviously was suffering from a pathological condition; then there were the "whining schoolboy, the lover sighing like furnace, the soldier seeking the bubble reputation in the cannon's mouth, and the justice in fair round belly"—all were pathological. Then, it would be remembered, "the sixth age slips into the lean and slippered pantaloon."

Dr. Dunlop, in reply, said that he realized he had a good deal to answer and would reply in a general way. In regard to the administration of glucose, he thought that Professor Lambie had summed up the matter well. It was his own practice not to give glucose until the blood sugar content had been reduced to 250 or 300 milligrammes per 100 cubic centimetres. Insulin was given with the object of reducing the blood sugar content; why give more sugar? As Professor Lambie had pointed out, clinical signs were important. Most authorities were agreed that much fluid given intravenously was dangerous. He had seen cases in which the circulation had been embarrassed. When he had talked of the administration of fluid he had meant rather subcutaneous, oral or rectal administration. He did not see any point in giving adrenalin, which would mobilize the glycogen and encourage the passage of more sugar into the blood, when the object of treatment was to reduce the blood sugar content. Possibly the adrenalin would help the circulation. It was of interest to note that some authorities were now giving ephedrine with the object of raising the blood pressure. Possibly with this agent the blood sugar would not be increased to the same extent. He did not think that anyone was prepared to declare that arteriosclerosis was caused by diabetes, but he wished to point out that there was more arteriosclerosis now and that there was a good deal of evidence to show that there was a distinct type of arterial degeneration that affected the young diabetic. As Professor Lambie had pointed out, the theories were at present largely speculative, but it was only by theorizing and presenting evidence that investigators would eventually arrive at the truth. The possible ætiological importance of syphilis had been mentioned; in the past he had arranged for many of his patients to be submitted to the Wassermann test; very few of them had reacted.

Dr. Shearman, in reply, remarked that diabetes had been cited as a possible cause of false positive reactions to the Wassermann test, but he had found at Sydney Hospital that the incidence of positive reactions among diabetics was low.

A meeting of the New South Wales Branch of the British Medical Association was held at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on August 27, 1931, Dr. George Bell, the President, in the chair.

Lesions of the Peripheral Nerves.

Dr. A. W. Campbell read a paper entitled: "Affections of Peripheral Nerves" (see page 544).

Dr. J. C. B. Allen read a paper entitled: "Affections of the Peripheral Nerves" (see page 546).

Dr. Allan S. Walker said that several conditions affecting the peripheral nerves were of special interest to physicians. Facial palsy, Bell's palsy, raised some important points. It should be noted that this was frequently associated with a demonstrable fibrositis in the peristyloid region, and this had a bearing on treatment. Similar lesions were also found sometimes in sciatica, in some cases there being an interstitial inflammation in the neighbourhood of the great sacro-sciatic foramen, and also in a less common state where an inflammatory disturbance in the sphenoidal fissure caused a temporary paralysis of the third, fourth, part of the fifth and the sixth cranial nerves.

The prognosis in Bell's palsy was the important thing for the patient, and the practitioner needed some general guide in the giving of an opinion. The slight cases were still obviously slight after, say, a week, movement was slightly impaired only, and rapid recovery ensued within a week or two. Where there was more severe loss of function, but still some ability to move the *orbicularis* and there was still excitability of muscles, recovery should take place without trouble in two or three months, but where the loss of power was still more marked, it was hard to forecast the time for recovery, as this might take many months and be associated with that curious phenomenon of facial palsy, contracture.

It would be of interest to hear the opinion of others on the subject of electrical treatment in cases of Bell's palsy. It was difficult to see what good it could do, and he could not find any adequate reason for its use.

As regards cervical rib, as Dr. Campbell had pointed out, the radiologist might not be able to help much, for the symptoms were frequently due to a fibrous band *et cetera*, and the presence or absence of an extra rib might be of no importance. Analogous to this was the class of case in which neuritic symptoms arose from pressure in *spondylitis deformans*. Here, too, the presence of bony outgrowths merely indicated the degree of demonstrable osseous change, for the nerve, if affected, was usually involved in a chronic inflammatory disturbance of the neighbouring interstitial tissue.

In sciatica Dr. Campbell had mentioned the classification of Sicard, and it would seem to be difficult but useful to try to distinguish which portion of the nerves was chiefly involved. He had certainly seen relief from the injection of some neutral fluid, such as normal saline solution, round the nerve, and would be glad to hear if any member had had personal experience with epidural injections in the cases of "high" sciatica.

The other forms of neuritis interesting to the physician included the various types of multiple neuritis and that due to diabetes. The latter appeared to be not uncommon in the mildest form, but severer grades were distinctly unusual. One of the latter had come under his notice in which there was severe and lasting paralysis of some of the forearm muscle groups. This patient was a musician,

but fortunately retained enough function to use of the hand of his bow arm.

In multiple neuritis it was always necessary to think of arsenic, though the proof was by no means simple in all cases. In one case under the care of a senior colleague the isolation of what appeared at first to be the *Bacillus typhosus* from the stool gave rise to considerable difficulty in diagnosis, the true cause being discovered to be arsenic.

He had seen recently a patient with a peripheral neuritis involving both lower extremities, in whom the occurrence of a zonal pain in the thoracic region suggested the diagnosis of a cord infection, after the style of a neuritic type of encephalomyelitis, but subsequent investigation proved this man to have an infective endocarditis; the neuritic symptoms here were apparently purely toxic.

Lastly, Dr. Campbell had given wise counsel on the subject of trigeminal neuralgia, particularly as regards the necessity of establishing a diagnosis of true *tic douloureux* before the adoption of any radical procedures. Attention had been drawn in recent literature to the risk of inviting bad results by improper selection of cases.

An emphatic protest should be made against wholesale extraction of teeth in these patients. Most unfortunates suffering from this malady were already edentulous when seen by the physician, and notwithstanding were still unrelieved. While it was undoubtedly important to eliminate such foci as truly septic teeth and disease of of the sinuses, it could be only harmful to remove from the patient his means of mastication and thereby seriously impair his nutrition. Of the radical operations, though this did not really come within the ambit of the physician, decided advance seemed to have been made in the procedure of partial trunk section behind the ganglion. But a plea should be made for earlier trial of alcohol injections. Surely in cases of the true major neuralgia of the fifth nerve it was better to perform this procedure much earlier in many cases instead of wasting valuable time in operations on teeth and sinuses where there was often very slender evidence of a pathological state. Such treatment would often be a great relief to doctor and patient alike.

Dr. D. J. Glissan expressed his interest in the papers. He referred to the comparative frequency with which the musculo-spiral and popliteal nerves recovered after suture and thought that the reason was an anatomical one. The actual fibres of supply came off fairly high up on the trunk of the nerve and the muscles were large and fleshy and concerned in the grosser movements. Dr. Glissan compared the nerves of the forearm and pointed out that in certain types of damage the axis cylinder had to travel a long way before it reached the muscles. In cases of injury to the ulnar nerve above the elbow, when suture was performed, it was found that the function of the *flexor carpi ulnaris* and the *flexor profundus digitorum* was usually recovered. But the intrinsic muscles of the hand were small muscles and could ill withstand lack of nerve supply. It was wiser to operate earlier in cases of ulnar and median damage, to see just what was the type of injury, rather than to wait. It was important to try to get the nerve supply back early to the small intrinsic muscles of the hand.

Dr. Glissan was glad to hear Dr. Campbell's protest against the loose diagnosis of neuritis; the general public appeared to love this diagnosis and all sorts of pain were thus described. The description was very often inaccurate and should be deplored. Dr. Campbell had catalogued a number of causes underlying so-called neuritis; Dr. Glissan wished to add another, namely, unreduced dislocation of the shoulder joint. He had seen this condition overlooked on several occasions and a diagnosis of neuritis of the shoulder made. In regard to brachial neuritis Dr. Campbell had referred to brachial neuralgia, but, in Dr. Glissan's opinion, there was also such a condition as brachial neuritis. In cases associated with trauma it had been his experience that when the patient complained of severe pain recovery was likely. It was in fact a favourable prognostic point if the patient complained of severe pain. However, when complete brachial plexus dissociation occurred, no pain was experienced. Dr. Glissan referred to a case of neuralgia associated with destruction of the acromio-clavicular joint. The clavicle had been pulled back and the patient had severe neuralgia associated with the radial nerve; operative restoration of the ordinary relationship between the acromion and the clavicle had led to recovery. Referring to multiple neuritis, Dr. Glissan recalled an interesting case which he had seen at the Children's Hospital. Dr. Campbell had also seen this case. Dr. Glissan had seen the patient while the condition was in its early stages; the child presented a picture suggestive of muscular dystrophy and hypotonia. Later neuritis developed. Dr. Glissan asked Dr. Campbell to tell them of this condition.

In regard to Dr. Allen's paper, Dr. Glissan said that he would like to take the opportunity of dissociating himself from the idea that electricity played any useful part in the recovery of paralysed muscles. If a facial paralysis recovered with electricity, it was in spite of rather than as a result of such treatment. He referred to a patient whom he had shown at Saint Vincent's Hospital. The median and ulnar nerves had been severed above the wrist and complete recovery occurred following splinting and reeducation and without the use of electricity in any shape or form.

An important sign when dealing with damaged nerves was formication; if it persisted only at the site of nerve injury, it might be taken as an indication of complete block and necessity for operation.

In regard to the pathology of damaged nerves, Dr. Glissan said that there was no more difficult problem than to decide what was best to do. They had before them the clinical and not the pathological picture of the damaged nerve. He would like to hear this point discussed.

As to facial paralysis, Dr. Glissan thought that as far as treatment was concerned, ordinary principles should be brought into play. They had to remember that the facial muscles were muscles of emotion and that smiling and scowling and other expressional activities placed a strain on them when they were damaged by paralysis. It was his practice to insist on the patient being isolated as far as possible from relatives and friends in order that these muscles should have adequate rest.

In conclusion, Dr. Glissan said that he would like to sound a note of warning with regard to those cases of ulnar palsy mentioned by Dr. Allen as appearing two or three weeks after injury to the elbow. He maintained that in the majority of such cases the damage to the nerve was coincident with the injury to the joint, and when symptoms were noted a few weeks later, it was very often because they had not been looked for at the time of the first examination. The bulk of elbow injuries occurred in children, and a small child would not tell the surgeon that it had numbness in the little finger. Ulnar nerve damage was easily missed and should always be looked for in damage to the elbow region.

Professor W. S. Dawson, in opening his remarks, paid tribute to Dr. Campbell's paper and to the skill with which he had presented a great deal of material without the intrusion of minor detail. Little could be added to such a paper. He wished, however, to refer to a couple of cases in which the diagnosis of multiple neuritis had been made, but in which pathological examination revealed a wider involvement of the nervous system than had met the clinical eye. The first was of the progressive type; the man had suffered for one year from numbness of the hands and from great general sensitiveness. Professor Dawson saw the man in the last couple of months of his illness. There was general emaciation and muscular wasting in the ulnar distribution of the left hand, also extreme tenderness of the nerve trunks. The second case was that of a woman whose lower limbs were affected. She had neuritis in association with tuberculosis—a rare condition according to some authorities. This woman was in an advanced stage of pulmonary tuberculosis. The lower limbs were wasted, the reaction of degeneration had been present and the tendon jerks absent. The lower limbs were extremely tender on palpation. In this case also there were widespread changes in the central nervous system, which Dr. Latham would be good enough to describe. Professor Dawson referred to neurotics and the difficulty of diagnosing their condition. They always provided a

snare and the medical practitioner had to beware of women who complained of agonizing pain in the face with numerous other symptoms without displaying the true picture of trigeminal neuralgia. The temperamental factor had to be recognized.

Brachial neuritis often occurred in individuals with a poor stance, laxity and poor tone of the shoulder girdle muscles, thus rendering the nerves liable to irritation. In Scotland, in spite of the national beverage, alcoholic neuritis was a rare condition, but mental affections from alcohol were far from uncommon.

DR. OLIVER LATHAM spoke of the two cases (mentioned by Professor Dawson) that had been diagnosed as peripheral neuritis. In both of these cases there were found a number of anterior horn cells showing distinct degeneration. In the instance of the male patient who had been under observation for a year, and in some of the cases which proceeded to a fatal conclusion, there was greater involvement of the central nervous system than of the peripheral nerves; there were found the common signs of a chronic or subacute encephalitis. The woman suffering from infectious disease had a toxic neuritis; the horn cells became affected, but the neuritis was toxic.

Professor Dawson and he, in going over cases in which involvement of the peripheral nerves only was suspected, found at times widespread affection of the central nervous system. It was rare to find purely local lesions; especially was this the case when the ætiology was of an infectious nature.

DR. N. D. ROYLE said that he had investigated various treatments for paralysed muscles, but that, apart from splinting and passive movement, none appeared to be of any value. He could not agree with Dr. Allen when he advocated electrical treatment. It had been shown several years before by Langley that paralysed muscles were in a state of continual fibrillation or fine movement. Electrical stimulation only increased that movement, caused overwork of the muscle and led to further wasting. Dr. Allen spoke of degeneration following stretching, but Dr. Royle maintained that intermittently stretched muscle recovered better than muscle that was not stretched; the stretching, however, must not be constant. It was absurd to speak of tone in paralysed muscles; paralysed muscles had no tone. As to Bell's palsy, he found that recovery occurred on an average after ten days' treatment. He treated the condition by massage of the neck from occiput to chin. He was interested in the view that fibrositis of the stylomastoid foramen was a factor in facial paralysis, and in fact, holding this view, he treated old cases with diathermy. As regards splinting of the face, Dr. Royle did not think that splinting in an angle of the mouth was adequate, because of the movement of the eye. He preferred to lift the whole side of the face and keep it in position by adhesive strapping. Ballance's experiments had been mentioned, but Dr. Royle did not think that they could credit much that Ballance said; he had during his recent visit put forward doubtful suggestions and had made what appeared to be inaccurate observations.

Dr. Royle also pointed out that maternal birth palsy was not due to the pressure on the head of the infant; it was far more probably due to rotation of the pelvis at the sacro-iliac joints.

Referring to Dr. Walker's mention of cervical rib, Dr. Royle said that there was one test of pressure on the brachial plexus when cervical rib could not be demonstrated radiologically, and that was to make the patient put his head back. The pulse in the arm disappeared from pressure of the *scalenus anterior* muscle and the removal of the *scalenus anterior* was thus indicated.

In conclusion, Dr. Royle quoted an instance in his experience of trigeminal neuralgia. The patient had all the signs except that of age—pain in the face, watering of the eye *et cetera*. Dr. Royle had divided the sympathetic trunk of the neck and the pain had disappeared. The pain was due to vaso-constriction of the trigeminal ganglion. If vaso-dilatation was produced by section of the sympathetic trunk the patient got relief from the condition.

Dr. Campbell, in reply, wished to thank particularly Dr. Walker, Dr. Glissan and Professor Dawson for their kind remarks touching his paper. In reply to Dr. Glissan's question concerning the case of multiple neuritis following measles which he saw at the Children's Hospital, Dr. Campbell said that he did not remember the case very well, but he had the impression that it was one of encephalomyelitis following measles rather than multiple neuritis. The brain was affected more than the peripheral nerves. He thanked both Dr. Glissan and Dr. Walker for the remarks they had made complementary to his paper, such as that it was possible to get cervical rib without signs of cervical rib and *vice versa*. Dr. Campbell did remember well a case of supposed cervical rib, and this would support what Dr. Royle had said. He had suggested to Dr. Wade that the *scalenus anterior* muscle should be divided. The result was satisfactory.

Dr. Walker had asked if he had had any experience of epidural injections. Dr. Campbell had had such experience. It was his custom to flood the sciatic nerve with normal saline solution, but in some cases he had in addition injected the solution into the epidural space. He could not, however, speak either favourably or unfavourably of the procedure.

In regard to Dr. Latham's observation, Dr. Campbell thought that if he had examined the peripheral nerves he would have found degeneration in them. Dr. Latham's comment on the pathology of the condition had been very interesting. It would be well if they could get closer to the causation of these conditions. Dr. Campbell had seen cases of encephalomyelitis of the neuritic type. If future cases came along it would be worth while to endeavour to reproduce the disease by injection into a monkey.

Dr. Allan, in reply, regretted that he had raised such an interesting and debatable point about electrical treatment in the stimulation of muscle. In spite of what Dr. Royle had said, he thought that it had a definite place in reeducation and in increasing the power of the muscles. In regard to Dr. Glissan's remarks about the ulnar nerves, Dr. Allen agreed that many of the early cases occurred at the time of the injury, but he was always careful to note the ulnar distribution, and he did think that there were a few such conditions manifested at the end of a few weeks after the injury. In speaking of early and late palsies, by "late" palsies Dr. Allen denoted those that occurred after some years; so that "early late" would refer to the early stages of a condition occurring after some years. In speaking of the musculo-spiral nerve, Dr. Royle had misunderstood him; he had said in the middle of the arm and not of the forearm. Dr. Allen had been interested in Dr. Royle's remarks about cervical sympathectomy in trigeminal neuralgia; he thought that this condition had much in common with causalgia.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Cramp, Cameron Oliver, M.B., B.S., 1930 (Univ. Sydney), 13, Birriga Road, Woollahra.

Dunstone, Reginald Murray, M.B., Ch.B., 1931 (Glasgow), c.o. Y.M.C.A., Pitt Street, Sydney.

Kennedy, Richard Thomas, M.B., B.S., 1929 (Univ. Sydney), 437, Marrickville Road, Dulwich Hill.

Shorter, Alan Ashton, M.B., 1930 (Univ. Sydney), 133, Middle Harbour Road, Lindfield.

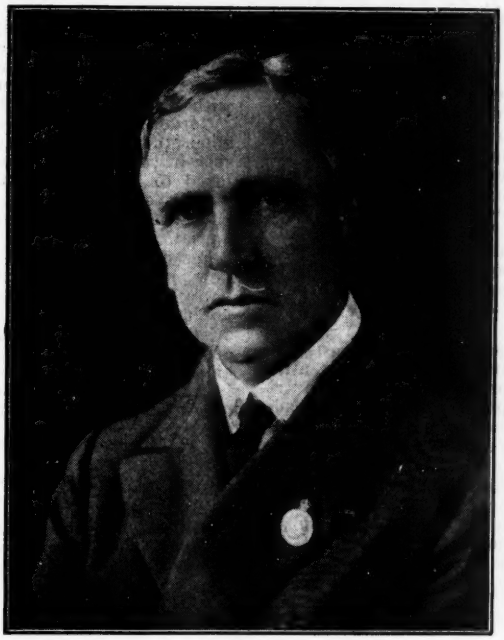
Obituary.

ROBERT GORDON CRAIG.

DR. ROBERT GORDON CRAIG, whose death was announced in a previous issue of this journal, was recognized throughout the Commonwealth of Australia as a conscientious and reliable surgeon, his fame had spread beyond its coasts

and he had many of the qualities which endear men to their fellows and which are remembered by those who follow. Gordon Craig made a name for himself, but he also did much for surgery—he did what few medical practitioners do, he gave back to the world of medicine some of the tangible wealth that he had won so that younger men might find the path easier than he found it.

Gordon Craig was born at Ardrossan, Scotland, on May 24, 1870. His father, Captain Robert Craig, came to Australia in 1877 as Marine Superintendent in Sydney for the Eastern and Australian Steam Navigation Company. The boy Robert followed his father in 1878 and had a stormy and adventurous journey in the ship *Cairnbulg*. The journey lasted from May till September and it seemed at one time as though the voyage would not be completed. The ship encountered a hurricane in the Indian Ocean and was dismasted; jury masts were rigged, and another hurricane was met on the Australian coast before Sydney Harbour was finally reached. After his arrival Gordon Craig attended Sydney Grammar School for a period. At the age of thirteen he was taken back to Scotland and put to school at George Watson's College in Edinburgh. He remained here for two years and then returned to Sydney. Although he did not distinguish himself at school, his masters always reported favourably on his ability. In 1888 he matriculated and passed to the University of Sydney. After the completion of one year in the Faculty of Arts he contracted enteric fever in a severe form. It was thought advisable that he should not resume his studies immediately, and he served for twelve months in the office of a shipping firm in Sydney. He then became a medical student. He gained an honours pass in his first year. In each of his subsequent years he topped his year; he won the John Harris Scholarship in his third year and won the University Medal for general proficiency throughout his course. He graduated Bachelor of Medicine and Master of Surgery in 1894. During his undergraduate life he took an active part in sport; he was champion long distance runner of his day and was awarded his "blue" for athletics.



After graduation Gordon Craig became Resident Medical Officer at the Royal Prince Alfred Hospital. He held this position for one year and then succeeded to the practice of the late Dr. Service at King Street, Newtown. In 1901 he was appointed Honorary Assistant Surgeon at the Royal Prince Alfred Hospital. He soon gained the recognition of the other members of the staff as a man of judgement and a careful operator. He was consistently careful of his work as a teacher of students and went to infinite pains to make them understand the reasons for what he did and to explain how he did it. About this time the late Critchley Hinder introduced cystoscopy to the Royal Prince Alfred Hospital. Gordon Craig was impressed with this kind of examination, and from this onwards his interest in endoscopic methods of examination and in genito-urinary surgery increased. It was not till much later, when he had devoted himself entirely to surgery and had held the position of honorary surgeon for some years, that he determined to devote his whole time to genito-urinary surgery. He founded the Department of Urology at the Royal Prince Alfred Hospital by a gift of £20,000, with the object of furthering urological research and making possible the appointment of Fellows in Urology. Important work has been carried out at this department, and its results have been published from time to time in other journals.

During the Great War Gordon Craig offered his services. In 1915 he was placed in command of the Australian Hospital Ship *Karoola*, and took great pride in organizing the unit and in helping to arrange the necessary alterations to the ship. After the evacuation of Gallipoli he was recalled and took up his duties at the Royal Prince Alfred Hospital and at the Prince of Wales Hospital, Randwick.

Gordon Craig was one of those who evinced an early interest in the College of Surgeons of Australasia (now the Royal Australasian College of Surgeons). He was a foundation Fellow and a member of the Council; he was also a member of the Credentials Committee of the College for New South Wales. He was a member of the Editorial Committee of *The Journal of the College of Surgeons of Australasia*. He made provision for the College in his will; it will benefit ultimately to the extent of £40,000. The sum bequeathed to the College is to be held in trust; one half of the income is to be devoted to the promotion of research in surgery and the other half is to be employed in assisting young graduates of exceptional ability and promise to undergo "the adequate post-graduate education which is an essential part of the training of a surgeon."

Among Gordon Craig's other activities must be remembered his work at the Royal Alexandra Hospital for Children and his membership of the Editorial Committee of *The British Journal of Urology*. He was an Honorary Fellow of the American College of Surgeons.

The British Medical Association claimed much of his attention. He was for many years a member of the Council of the New South Wales Branch and was President in 1917-1918. He was President of the Section of Surgery of the First Session of the Australasian Medical Congress (British Medical Association). At meetings of the Association he read papers which were published in this journal. The papers without exception bore the stamp of earnest endeavour.

Gordon Craig had a happy home life. He had as hobbies golf, yachting and caravaning. The last months of his life, restricted as they were by his first illness, must have been particularly trying to a man of his boundless energy. He undoubtedly left his mark in the world of medicine and a name which will be honoured.

Dr. H. H. Bullmore writes:

It would be a commonplace to say that the niche which Gordon Craig filled in the community, especially in the medical community, seems very empty now. Seldom has one been so mourned by so many in all walks of life, in all grades of society. The members of his profession miss him the more because of the reliance they knew they could place in his judgement and operative work. Gordon Craig's aim was efficiency; his dominant characteristics, thoroughness and enthusiasm, were just the qualities required to attain his objective. We, his colleagues, knew that anything Craig undertook to do was done thoroughly, the details were attended to as meticulously as the more important steps in an operation or in the making of a diagnosis. Craig rarely had an idle moment and could hardly have coped with the amount of work he managed to get through but for his capacity for organizing. It

was this power of his that made the hospital ship *Karoola*, of which he was in charge during a part of the war, such an exceedingly efficient unit.

He so organized his work as to allow him to deal with a regular clinic in his private practice and also to find time to engage in many non-professional activities. The Urological Department of the Royal Prince Alfred Hospital is an evidence of his belief in organization and of his foresight, as well as being a monument of philanthropy and patriotism. It was a magnificent expression of gratitude to the institution to which he felt he owed so much. Few have had the power or inclination to make such a return.

It has been the lot of few men to have become so successful in so many spheres of life as Craig and at the same time to preserve the respect and affection of all, both inside and outside their own profession.

Gordon Craig was not a genious in any sense of the word, but he was an outstanding man; he took a prominent position in any activity he engaged in. As a general practitioner he was most successful, as a general surgeon he was as successful, and when later he devoted himself particularly to urological surgery, his work was of the highest order. He spared neither trouble nor expense to attain a thorough knowledge of the subject in which he was interested. Every five years or thereabouts he visited America and Europe to inspect the clinics in which he was interested. During his trips he gave up very little time to pleasure, but worked hard and long hours.

He engaged in many non-professional activities, of which few knew, for Craig was a community man. He was on the board of some large business undertakings as well as being an active member of some of the charitable undertakings initiated by the Rotary Club, of which he was a member. At the time of his death he was Vice-Captain of the Australian Golf Club. He was an enthusiastic and very good golfer. His happiest days were spent with Mrs. Craig on their frequent caravan trips. They had journeyed through most parts of New South Wales and loved every moment of their holidays.

For some years he had been particularly interested in the work being carried out by the Council for Scientific and Industrial Research in the problems of animal nutrition and disease, and pasture improvement, and had made up his mind to divert some of his energies to these problems when the time came to retire from the Royal Prince Alfred Hospital.

Last year he acquired a property admirably suited to his needs, and there he lived for the last three months of his life recuperating after the long rest in bed subsequent to the coronary occlusion which occurred in December of last year. It was a hard matter for one of Craig's temperament to restrain his physical activities during this time. That he was supremely happy in his new surroundings during this three months is a consolation to all his friends.

Dr. C. Bickerton Blackburn writes:

I first came into close personal contact with Gordon Craig some thirty years ago when he was carrying on an enormous family practice in Newtown. I acted as his *locum tenens* on several occasions when he took his annual holiday, and so had abundant opportunities of observing the confidence and affection he inspired in all types of patients. There was scarcely a case of serious illness in the whole district that he did not see, for if he was not the family attendant, he was called in as a consultant. His powers of endurance were amazing, and scarcely a night passed without his being called out of his bed two or three times. He had a little window on the floor above his front door and he used to tell many amusing stories of early morning interviews that took place from this vantage point. He was much more than a doctor to the big industrial population, and all sorts of problems used to be thrashed out in his surgery, ranging from the desirability of purchasing a horse by the local cabman to the arranging of a much needed marriage with a jibbing swain. In after years it was a delight to his friends to draw him on to recount some of the droll experiences of those early days.

Physical fitness was almost an obsession with him, and he somehow found time to become a first class golfer. He was passionately fond of sailing and was recognized as one of Sydney most expert yachtsmen. For many years he spent all his week-ends in his famous boat, *Heather*, in which he took part in many thrilling races.

Craig always had a surgical career in view, and when still in general practice made time to see much of the surgery at the metropolitan hospitals. The habit thus formed of watching others at work he continued throughout his life and always maintained that there was much more to be learned from watching a man do an operation than from reading a description of how he did it. This led him to pay periodical visits to Europe and America, where he spent most of his time at surgical clinics. The same faith in personal contact brought him to every Australasian Medical Congress, where he expected to learn more from informal gatherings than from the set pieces, and he would sit till the small hours surrounded by kindred spirits picking their brains and giving freely in return. It was a delight to watch him in an argument, keen and enthusiastic and most tenacious of his point of view, yet always ready to give the other side a fair hearing.

In big things he really liked to get the view of others before making up his mind, and long before he established the urological foundation at the Sydney University which bears his name, he discussed every aspect of the question with his intimate friends.

Gordon Craig was proud of his Scotch nationality, and when there was anything Scotch in the atmosphere his speech would unconsciously take on a broader accent. He loved to hint at a vein of Scotch thrift and caution in his disposition, but was actually extremely open-handed and generous, and quite incapable of resisting any genuine appeal.

His domestic life was serenely happy and he well knew how much he owed to his wife's unselfish devotion. Away from his work he had no plans in which she did not figure, and his one idea of a holiday was to take her off into the bush on camping trips. For this purpose he designed a wonderful motor caravan fitted with comfortable beds, a kitchenette and every possible convenience, in which, like Gipsies, they roamed at one time or another over almost every part of the State.

THOMAS HOWARD MORGAN.

We regret to announce the death of Dr. Thomas Howard Morgan, which occurred at Brisbane, Queensland, on October 17, 1931.

JOHN MONASH.

Reference is made on page 555 to the death of Sir John Monash. On page 567 is reproduced a picture of the memorial sent on the occasion of the funeral from the officers of the Australian Army Medical Corps of New South Wales. The tablet, which is the work of Wunderlich, Limited, is a fine example of the craftsman's art. It weighs thirty pounds and measures 22½ by 18 inches.

Correspondence.

ORTHOPÆDIC SURGEONS.

Sir: I confess the remarks of Dr. Corlette which prompted my protest in your issue of September 12 had surprised me—not "hurt my feelings"—as I thought them made by one in whom the desire for scientific accuracy was paramount. His letter published in the journal of October 10 dispels that idea, for he excuses the statements I challenged on the grounds of rhetorical expediency. "Rhetorical expedients" for the purpose of "causing people

to take notice" have never been considered quite the thing in medical gatherings—so I have been taught—however badly one "wanted notice to be taken." I wonder how much of his letter of October 10 is "rhetorical expedient" and how much his belief.

Does he really believe that a surgeon, because he pays particular attention to one branch of his profession, ceases by so doing to have any understanding of the problems of that branch? Why does he so rage against "principles"? Surely he must have some himself? Are they too "empirical formulas"? When he suggests that a worker with a pain in his back might do better in the hands of "faith healers" *et cetera* than in those of orthopædic surgeons, is he in earnest or is he again in the throes of rhetorical expediency?

His appreciation of orthopædic principles would seem to be even less than was apparent from his original remarks (that is, those protested against), since he appears to believe the "formula" for a pain in the back to be "plaster jacket."

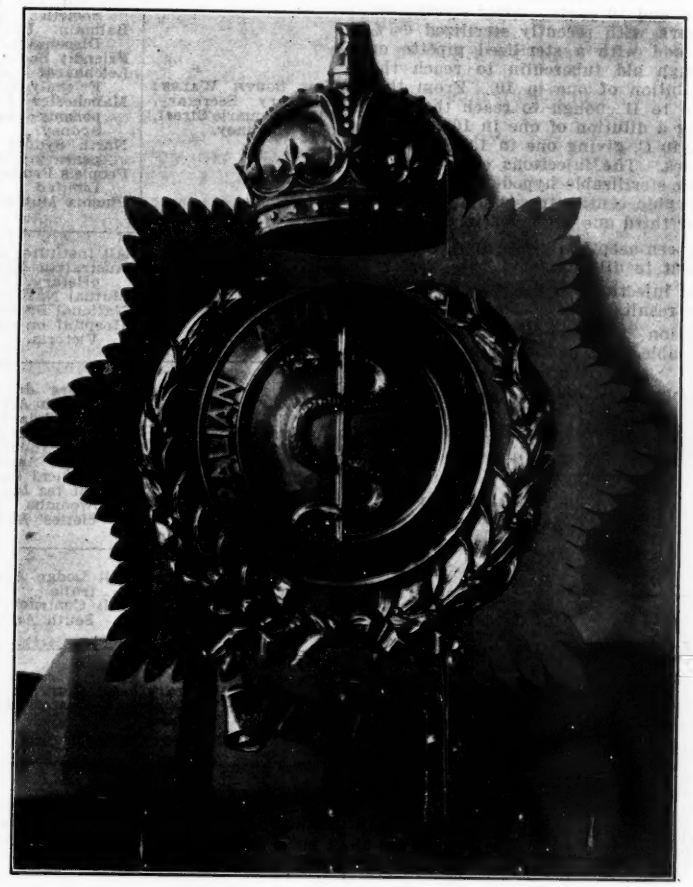
To again quote his own words: "if he can but unbandage his eyes and look," he may find he has built up his criticism, "scepticism (and worse)" on foundations of misconception and misunderstanding—that is, if his spoken and written word represents his belief as regards these "principles."

Orthopædic surgeons are fully alive to the "psychological handicaps" from which many patients suffer. Many of these would not be so heavy had previous treatment been guided by sound principles. Opinion may differ as to what constitutes good or bad taste. My protest was against a member of the Section of Surgery attacking the alleged methods of members of the Section of Orthopædics, to the extent of claiming that an orthopædic surgeon must die in order for his patient to recover, at a combined meeting of those sections (with radiology), the object of which was presumably calm, scientific discussion. And this in the presence of a distinguished lay visitor, Judge Perdriau. My opinion on this point remains unaltered.

Yours, etc.,

JOHN HOETS.

143, Macquarie Street,
October 19, 1931.



Memorial to Sir John Monash.

THE DIAGNOSTIC USE OF TUBERCULIN.

SIR: After reading with great interest the instructive article on the diagnostic use of tuberculin appearing in the issue of October 17, might I be allowed to record some conclusions reached as the result of using this test in private and hospital practice for thirty years.

Subcutaneous tuberculin injection as a diagnostic procedure differs from the cutaneous, intradermal or ophthalmo reaction in that in addition to the local there is a general and focal reaction.

The focal reaction is useful in cases of doubtful laryngeal, glandular or joint conditions and in determining the cause of pleural and obscure persistent pain. Occasionally only does it locate an undetected pulmonary focus. Sometimes it reveals a quite unsuspected lesion in bone or joint.

The reaction depends on an acute inflammatory response at the focus of disease which, if excessive, may undoubtedly lead to exacerbation and activation. Though uncommon, I have seen a few cases where the reaction was definitely detrimental. It is necessary, therefore, to avoid excessive focal reactions and to be content with the minimum general reaction which is consistent with diagnostic certainty.

Most patients, where diagnosis is sought, are not completely afebrile, presenting temperatures from 99° to 99·5° or even 100° F. Higher temperatures preclude the use of this test.

Where the temperature ranges below 100°, it is necessary to take four-hourly observations for at least forty-eight hours prior to the test, preferably longer. The usual practice is to consider a rise of one and a half degrees above the highest control reading a positive reaction. The reaction rarely commences within twelve hours of injection, practically never within eight. It may reach its maximum and start to fall within four hours of onset. Hence the method adopted at the Royal Prince Alfred Hospital, of giving the injection at 10 p.m., taking temperature at 2 a.m., 6 a.m., then two hourly till a rise of temperature is noted, then hourly till it falls. This precludes the chance of missing a reaction and so subjecting the patient to an unnecessarily large dose. It involves the patient resting continuously under skilled supervision as far as taking temperatures is concerned during the test. To inject the test at the dispensary or consulting room and send the patient off to take his temperature four hourly when awake seems to me to involve unjustifiable risk. He may easily miss or sleep through a mild reaction and then be subjected to an unnecessarily large dose. It is such procedures that earned the reaction its bad name in the past and provokes in the Extra Pharmacopœia the statement that "in human beings this test is not usually considered safe." Done under careful supervision, it is practically free from danger.

As regards test solutions my experience is that dilutions ready made by manufacturing firms are often inert and cause numerous mistakes. A dilution of one in ten keeps for months. Higher dilutions should not be kept longer than a week or ten days.

Dilutions can be readily made by taking three small, narrow bottles, A, B, C, fitted with rubber corks. In each, nine measures of, say, 15 minims, that is 135 minims, of water are placed. The amount is immaterial as long as the multiples are right. At the upper level of the fluid a small strip of "ZO" strapping is attached so that its upper edge is level with the bottom of the meniscus. Fifteen minims are added and the level similarly marked. With a triangular file mark on the glass the levels as shown by the two pieces of strapping. You have now three bottles marked at 9 and 10 parts. After sterilizing, fill each to the lower mark with recently sterilized 0·5% phenol solution. To A add with a sterilized pipette or hypodermic syringe enough old tuberculin to reach the upper mark, giving a dilution of one in 10. From this bottle, after shaking, add to B enough to reach the level of the upper mark, giving a dilution of one in 100. From B fill to the upper mark in C, giving one in 1,000. Label the dilution on the bottles. The injections can be made with an all glass ordinary sterilizable hypodermic syringe, giving as first dose one cubic centimetre (17 minims) of C, for second 0·5 of B, for third one cubic centimetre of B.

If your dilutions have been aseptically prepared, there is no need to boil subsequent to dilution.

Take care not to repeat injections in the same area or a false local reaction may result.

Covering the inoculation puncture with plaster is unnecessary and inadvisable. It occasionally produces local irritation.

In the diagnosis of pulmonary disease tuberculin has been almost completely superseded by the radiographic screen and film. It still has a useful field if handled with care and discrimination.

Yours, etc.,

Sinclair Gillies.

185, Macquarie Street,
Sydney,
October 21, 1931.

Diary for the Month.

Nov. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 5.—South Australian Branch, B.M.A.: Council.
Nov. 6.—Queensland Branch, B.M.A.: Branch.
Nov. 10.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 11.—Victorian Branch, B.M.A.: Branch.
Nov. 12.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 12.—Victorian Branch, B.M.A.: Council.
Nov. 13.—Queensland Branch, B.M.A.: Council.
Nov. 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 25.—Victorian Branch, B.M.A.: Council.
Nov. 26.—South Australian Branch, B.M.A.: Branch.
Nov. 26.—New South Wales Branch, B.M.A.: Branch.
Nov. 27.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser" page xiii.

Launceston Public Hospital, Tasmania: Medical Superintendent.

Royal Hospital for Women, Sydney, New South Wales: Resident Medical Officer.

Sydney Hospital, Sydney, New South Wales: Honorary Gynæcological Surgeon.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
South Australian: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor," The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £1 for Australia and £2 5s. abroad *per annum* payable in advance.